A Dissertation on

"A STUDY OF THE CLINICAL SPECTRUM OF POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME IN A TERTIARY CARE HOSPITAL"

Submitted to

THE TAMILNADU
DR. M.G.R. MEDICAL UNIVERSITY

In partial fulfilment of the requirements
For the award of degree of

D.M. (Branch - 1)
NEUROLOGY

GOVERNMENT STANLEY MEDICAL
COLLEGE & HOSPITAL
THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY
CHENNAI, TAMILNADU

AUGUST 2014

CERTIFICATE

This is to certify that the dissertation entitled **"A STUDY OF THE CLINICAL SPECTRUM OF POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME IN A TERTIARY CARE HOSPITAL**" is a genuine work done by Dr. R. KALPANA for the partial fulfilment of the requirements for D.M. (Neurology), examination of the **The Tamilnadu Dr.M.G.R. Medical University** to be held in August 2014, under the able guidance and supervision of **Prof.Dr.S.GOBINATHAN, M.D.,** D.M.,(Neurology), Professor and Head, Department of Neurology, Government Stanley Medical College and Hospital, Chennai.

Prof. Dr.S.GOBINATHAN
M.D., D.M, (Neurology)
Prof & Head of Neurology
Govt Stanley Medical College &
Hospital, Chennai – 600 001

Prof. Dr.AL.MEENAKSHI SUNDARAM,
M.D., D.A.,
Dean
Govt Stanley Medical College &
Hospital, Chennai – 600 001

DECLARATION

I, **Dr.R.KALPANA,** Solemnly declare that the dissertation **"A STUDY OF THE CLINICAL SPECTRUM OF POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME IN A TERTIARY CARE HOSPITAL**", is a bonafide work done by me during the period of January 2012 to January 2014 at the Government Stanley Medical College and Hospital, Chennai under the expert supervision of **Prof. Dr. S. GOBINATHAN., M.D, D.M., (Neurology)**, Professor and Head, Department of Neurology, Government Stanley Medical College and Hospital, Chennai.

This thesis is submitted to **The Tamilnadu DR.M.G.R. Medical University** in partial fulfilment of the requirements for D.M. (Neurology), examination to be held in August 2014

Chennai **Dr. R. KALPANA**

Date:

ACKNOWLEDGEMENTS

I wish to express my sincere thanks to **Prof.Dr.AL.MEENAKSHI SUNDARAM, M.D, D.A.,** Dean, Government Stanley Medical College and Hospital for having permitted me to utilize the facilities of the hospital for the conduct of the study.

My heartfelt gratitude to **Prof.Dr.S.GOBINATHAN, M.D., D.M.,(Neurology),** Professor and Head, Department of neurology, Government Stanley Medical College Hospital for his guidance, motivation, valuable suggestions, expert supervision and for making all necessary arrangements for conducting this study.

I express my sincere gratitude to **Dr.S.SAKTHI VELAYUTHAM, M.D., D.M., (Neurology)**, my Assistant Professor, for his valuable guidance and the suggestions throughout the period of this study.

I express my sincere gratitude to **Dr. P. R. SOWMINI, M.D., D.M., (Neurology)**, my Assistant Professor who had evinced constant and keen interest in the progress of this study and was instrumental in successful completion of the study.

I express my sincere gratitude to **Dr.K.MALCOLM JEYARAJ, M.D., D.M., (Neurology)**, my Assistant Professor for his valuable guidance, keen interest and suggestions throughout the period of this study.

I would like thank to **Prof. Dr. C. AMARNATH, M.D., F.R.C.R., M.N.A.M.S.,** Professor and Head, Department of Radiology, Government Stanley Medical College Hospital, who offered guidance and radiological diagnosis throughout the period of study

My sincere thanks to all the fellow postgraduates in the department of neurology who helped me during this study.

I thank my husband **Dr.V.S.SENTHIL KUMAR, M.D., (Anaesthesiology),** for helping me in compiling this thesis.

I thank the staff nurses and M.R.I Technicians, Government Stanley Medical College Hospital for their cooperation and assistance.

I owe my gratitude for all the patients and their family included in this study, for their whole hearted cooperation, without them the study could not be possible.

CONTENTS

Sl.No.	Title	Page No.
1.	INTRODUCTION	1
2.	AIM OF THE STUDY	14
3.	REVIEW OF LITERATURE	15
4.	MATERIALS AND METHODS	20
5.	RESULTS	23
6.	SUMMARY OF THE RESULTS	43
7.	DISCUSSION	45
8.	CONCLUSION	49
	ANNEXURES	
	A. BIBLIOGRAPHY	
	B. MASTER CHART	
	C. PROFORMA	
	D. ETHICAL COMMITTEE APPROVAL LETTER	
	E. TURNITIN SNAPSHOT	
	F. PATIENT INFORMATION SHEET	
	G. INFORMED CONSENT FORM	

INTRODUCTION

Hinchey and co workers in 1996 first described a clinico-radiologic entity, in which patients presented with a symptom complex of sudden onset headache, altered sensorium, visual symptoms and seizures that, was later termed as Posterior Reversible Encephalopathy Syndrome (PRES).

Posterior Reversible Encephalopathy syndrome is a syndrome in which abrupt rise in blood pressure or use of certain drugs or sepsis leads to sudden onset of headache, altered sensorium, visual symptoms and seizures.

These symptoms occur in various combinations or sometimes in isolation. Hence a high index of suspicion to diagnose this syndrome is essential whenever a clinical setting that can predispose to Posterior Reversible Encephalopathy Syndrome exists.

Cases of Posterior Reversible Encephalopathy Syndrome are reported from age 4 up to 90 years of age, however most cases occur in the young and the middle age group (14).

There is a high female preponderance for Posterior Reversible Encephalopathy Syndrome as the cause of the disease is often pre-eclampsia or eclampsia. Other common co morbidities are renal

failure, systemic hypertension presenting as accelerated hypertension, bone marrow transplantation and solid organ transplantation (1,14)

Clinical symptoms resolve within few days to weeks with appropriate treatment. Though the term Posterior Reversible Encephalopathy Syndrome denotes reversible encephalopathy, irreversible neurologic deficit occurs if prompt identification of the disease and early appropriate intervention is not done.

Ischemic infarcts and hemorrhages are the complications of posterior reversible encephalopathy syndrome that lead to persistent neurological deficit.

PRES: Pathophysiology

The Pathophysiology of Posterior Reversible Encephalopathy Syndrome is still elusive. Two theories have been postulated – the hyperperfusion theory and the hypoperfusion theory.

PRES: Pathophysiology in patient with hypertension

Systemic Hypertension is a common medical problem worldwide. It is the commonest cause of Posterior Reversible Encephalopathy Syndrome.

The hyperperfusion theory is implicated here. The rapidity of rise of blood pressure is more important than the absolute value of blood pressure in the development of Posterior Reversible Encephalopathy Syndrome.

The exact pathophysiology of Posterior Reversible Encephalopathy Syndrome is still undetermined. It is postulated the raise in blood pressure causes vasodilatation, hyperperfusion, blood-brain-barrier breach and extravasation of intravascular fluid into the interstitium leading to vasogenic cerebral edema and Posterior Reversible Encephalopathy Syndrome.

There is a protective, compensatory mechanism for this excessive vasodilatation, namely the cerebral autoregulation mediated by the sympathetic nerves. This autoregulation maintains constant blood flow to the brain in spite of blood pressure fluctuations.

These sympathetic nerves mediate vasoconstriction and is effective in preventing excessive vasodilatation and cerebral edema formation. There is a rich sympathetic innervation of the internal carotid and anterior cerebral circulation where as the posterior circulation, the basilar artery and its branches have only sparse sympathetic innervation.

Hence when there is an abrupt raise in the blood pressure in the less protected posterior circulation there is vasodilatation, extravasation of fluid into the interstitium causing vasogenic edema and Posterior Reversible Encephalopathy Syndrome.

When the mean arterial pressure (MAP) is within the range of 60 - 120 mmHg the cerebral auto regulation is intact, causing variable vasodilatation and vasoconstriction, thereby maintaining the cerebral blood flow at around 40 - 60 ml / min / 100 grams of brain tissue.

However when the mean arterial pressure exceeds the value of 170 mmHg the cerebral auto regulation is disrupted leading to vasogenic edema and Posterior Reversible Encephalopathy Syndrome.

During many instances, however, a smaller MAP increase of even a few mmHg as seen often in pregnancy induced hypertension can cause disruption of cerebral autoregulation.

PRES: Pathophysiology in patient without hypertension

The First description of this disease entity – Posterior Reversible Encephalopathy Syndrome in 1996 was in a patient being treated with cyclosporine.

The drugs implicated in causing PRES are mainly the immunosuppressant drugs- like cyclosporine, cisplatin, oxaliplatin, Tacrolimus. Several other drugs like α- interferons and erythropoietin, cytarabine, methotrexate, rituximab, linezolide, high dose corticosteroid therapy etc. are also implicated in causing Posterior Reversible Encephalopathy Syndrome. (14)

The mechanisms by which these drugs cause Posterior Reversible Encephalopathy Syndrome is multi factorial. They either cause drug induced hypertension and or endothelial damage.

PRES: Pathophysiology in patients with Sepsis

Sepsis causes Posterior Reversible Encephalopathy Syndrome by two mechanisms – endothelial dysfunction and microcirculation abnormality. Endothelial dysfunction can be due to direct effect of the pathogen or secondary to mediators of inflammation.

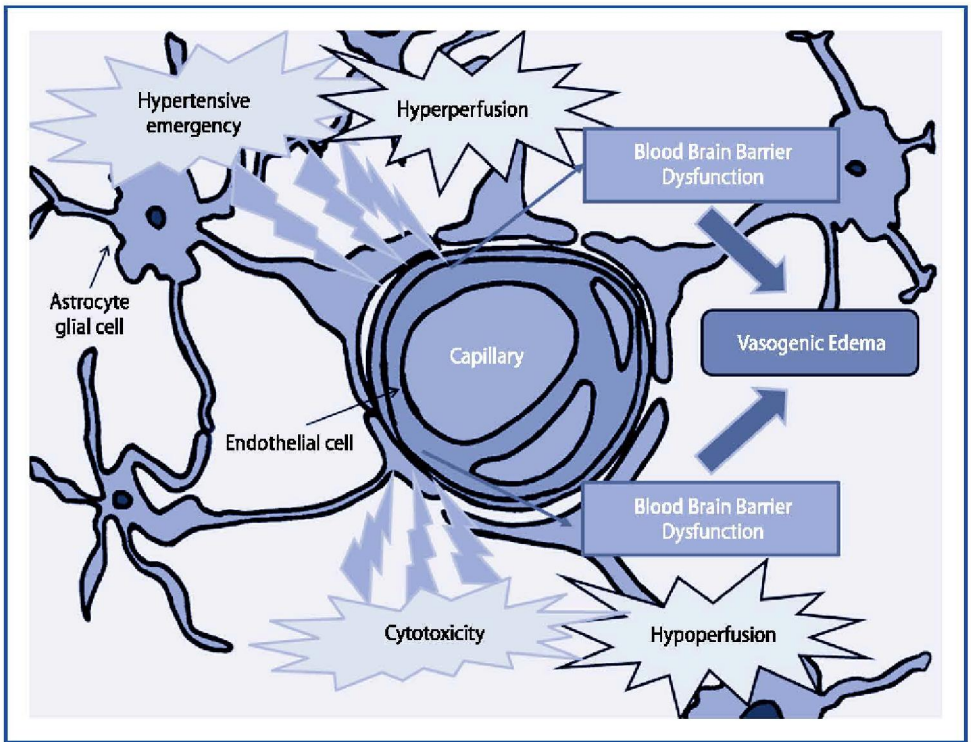
Microcirculation abnormality is due to the blockade of microvasculature by the leukocytes and inflammatory products. They block the microcirculation causing tissue hypo perfusion and also there is change in the vascular tone brought forth by the vasoactive substance released during inflammation.

This microcirculation changes in sepsis predispose to Posterior Reversible Encephalopathy Syndrome. Also infections like retro-viral infection (HIV) per se can precipitate Posterior Reversible Encephalopathy Syndrome, even when sepsis or septic shock has not occurred.

In patients of Posterior Reversible Encephalopathy Syndrome without hypertension i.e. PRES associated with infection, sepsis, certain drugs, endothelial surface antigens get up-regulated and there is release of endothelin which affects the vascular tone causing vasoconstriction and hypoperfusion. This hypoperfusion is followed by blood brain barrier dysfunction leading to vasogenic edema and Posterior Reversible Encephalopathy Syndrome. This is the cerebral hypoperfusion theory implicated in patients of Posterior Reversible Encephalopathy Syndrome without hypertension.

Recurrent Posterior Reversible Encephalopathy Syndrome has been reported in 6% of patients. (14)

Death following Posterior Reversible Encephalopathy Syndrome has been reported in up to 15% of the patients. (14)



This figure shows the two main hypotheses of posterior reversibnle encephalopathy syndrome (PRES) pathophysiology. One involves impaired cerebral autoregulation responsible for cerebral hyperfusion and blood brain barrier dysfunction. The other is related to cytotoxicity and involves endothelial dysfunction, blood brain barrier alteration and cerebral hypoperfusion. Under both hypotheses, the result of the cerebral blood perfusion abnormalities is cerebral vasogenic edema.

PRES and its complication – Pathophysiology

Cerebral infarction, hemorrhage and brain stem herniation are the irreversible complication of Posterior Reversible Encephalopathy Syndrome.

Cerebral infarction or cytotoxic injury occurs when the blood pressure is not controlled early and promptly – the endothelial dysfunction causes vasospasm leading to cerebral hypoperfusion, cytotoxic injury and infarction.

Reperfusion causes bleeding into the infarcted area causing hemorrhagic infarct. These infarcts and hemorrhagic infarcts are associated with cytotoxic edema (Direct damage to cells) adding on to the vasogenic edema (mediated by the extravasated toxic blood products) that occurs early in this disease entity – raising the intracranial pressure further and cerebral herniation , death can occur, if not treated promptly.

The Radiologic features of PRES

Vasogenic edema is the classical feature of Posterior Reversible Encephalopathy Syndrome. It commonly presents with bilateral symmetrical vasogenic edema typically involving the sub-cortical white matter, predominantly in the bilateral parietal occipital lobes – this is called the dominant parieto occipital pattern.

Sudden rise in the blood pressure exceed the auto regulatory capability of the vasculature of the brain causing regions of hyperperfusion (vasodilatation) and hypoperfusion (vasoconstriction) develops especially in the watershed zones and break down of the blood - brain barrier ensues with local extravasation of fluid and petechial hemorrhages.

Diffusion – weighted images (DWI) typically show iso-intense or hypo-intense lesions, with hyper intensity in the corresponding areas in ADC due to increase in apparent diffusion coefficient for water, indicative of vasogenic brain edema.

Other patterns are the holohemispheric water shed (anterior and posterior water shed) involving the frontal, parietal and occipital regions with less of temporal lobe involvement.

The Superior frontal sulcus pattern – area of edema that have patchy distribution along the superior frontal sulci in the frontal lobe. The parieto – occipital lobes may or may not be involved.

Partial expression of the three primary patterns with vasogenic edema involving both the parietal and frontal lobes but excluding the occipital lobe may be seen. Asymmetric involvement may also be noted. This is the Partial and or Asymmetric pattern.

Clinical Manifestation of PRES

The classical presentation of Posterior Reversible Encephalopathy Syndrome is altered sensorium, headache, visual symptoms and seizures in various combinations or sometimes in isolation as well. Hence a high index of suspicion is required to diagnose this syndrome as mentioned earlier.

Diagnosis of PRES

PRES is a clinicoradiological diagnosis.

Hence a clear knowledge, about the situations that could predispose to Posterior Reversible Encephalopathy Syndrome and the radiological patterns of PRES, is crucial to diagnose this condition.

T2FLAIR, ADC, DWI sequences are important in diagnosing PRES.

Treatment of PRES

General Measures:

Intensive care setting may be required in many cases. Most of the patients are hemodynamically stable but a few may require inotropic support. The patient may require endotracheal intubation and the need for

the same should be continuously assessed for upper airway protection in marked impairment of consciousness or resistant seizures.

Rapid induction with thiopentone or propofol with succinylcholine (exclude hyperkalemia) are good choices, if endotracheal intubation is done. Hypoglycemia should be checked and corrected. 100mg of thiamine should be given, simultaneously to hypoglycemia correction especially if there is B1 vitamin deficiency possibility like hyperemesis gravidarum.

Metabolic parameters should be closely monitored and particularly hypomagnesaemia should be promptly corrected.

Control of Seizure:

At the presentation of seizures, I.V lorazepam 0.1mg/ kg I.V. (maximum dose of 4mg) or diazepam 0.2 mg/ kg I.V. (maximum dose of 10mg) over 1 minute. Allow 5 minutes to determine whether seizures terminate, if no response repeat once.

If seizures persists, loading dose of phenytoin 20 mg/ kg I.V. at the maximum rate of 50 mg/ minute or phosphenytoin 15 – 18 mg phenytoin equivalent (PE)/ kg/ min I.V. at the maximum rate of 150mg PE/ min or phenobarbitone 20 mg/ kg I.V. at 60 mg/ min should be given.

Midazolam, propofol or thiopentone in titrated doses is needed in patients having refractory status epilepticus until the clinical seizure remits.

For patients showing electrical status epilepticus, titrated doses of Midazolam, propofol or thiopentone is given until burst suppression is obtained, this is followed by continuous infusion of the drug for at least 24 hours.

Hypertensive emergency management

Normalizing the blood pressure is not the aim here. Instead reducing the MAP by 20 – 25% within the first 2 hours and down the blood pressure to 160/100 mmHg within the first six hours is aimed at. More rapid or aggressive correction of blood pressure is not recommended because it can lead to cerebral hypoperfusion and cause infarct.

I.V. antihypertensives are needed many times. I.V. labetalol, nicardipine are appropriate choices if available. I.V nitroprusside, nitroglycerine can be used. Anti-edema measures like mannitol, furosemide may be needed.

Underlying cause correction

Etiological diagnosis of PRES should be identified early to allow prompt correction of the cause precipitating PRES. Blood pressure reduction, withdrawal of offending drug, termination of pregnancy – cesarean section, dialysis etc., may be required. Prompt treatment of the cause prevents irreversible complications and death.

AIM OF THE STUDY

To study the etiological, clinical and radiological profile of patients diagnosed to have Posterior Reversible Encephalopathy Syndrome.

REVIEW OF LITERATURE

Hinchey et al at New England Medical center, Boston, in 1996 first described a reversible disease entity in a series of 15 cases presenting with headache, altered sensorium, seizures, visual disturbance with findings of predominant posteriorly distributed leukoencephalopathy on brain imaging–he called this as reversible posterior leukoencephalopathy syndrome (RPLS)

In 2000, Sean O Casey proposed the more appropriate term of Posterior Reversible Encephalopathy Syndrome - PRES instead of RPLS. This term Posterior Reversible Encephalopathy Syndrome is more apt as there is no leukoencephalopathy or destruction of the white matter as such but rather a vasogenic edema in the subcortical white matter following encephalopathy.

Hinchey in N Eng J Med 1996 reported that “Of the 15 patients, 7 were receiving immunosuppressive therapy after transplantation or as treatment for aplastic anemia, 1 was receiving interferon for melanoma, 3 had eclampsia, and 4 had acute hypertensive encephalopathy associated with renal disease (2 with lupus nephritis, 1 with acute glomerulonephritis, and 1 with acetaminophen induced hepatorenal failure)”. He further documented that “12 patients had abrupt increases in blood pressure and 8 had some impairment of renal function”. The CT

and MRI imaging of these patients showed extensive subcortical white matter edema predominantly in the posterior cerebral hemisphere. These patients were “treated with antihypertensive medications, and immunosuppressive therapy was withdrawn or the dose reduced. In all 15 patients, the neurological deficits resolved within 2 weeks”.

The cause of PRES is multifactorial. The first description of Posterior Reversible Encephalopathy Syndrome was given in a patient receiving cyclosporine. By far the commonest causes are sudden elevation of blood pressure as seen in eclampsia, renal failure and usage of drugs like immunosuppressants.

Sioane et al. noted blood brain barrier abnormality at autopsy, in two patients who were bone marrow transplant recipients treated with cyclosporine. Others have suggested that the toxic effect of cyclosporine is seen only in patients having pre- existing blood brain barrier breach. This hypothesis was commonly seen in liver transplant recipients receiving cyclosporine or Tacrolimus already had more episodes of encephalopathy prior to transplantation.

Tdileman et al also confirmed that cyclosporine toxicity occurs only in patients with already pre existing blood brain barrier damage, secondary to infection.

Bartynski et al in 2007 reported 136 patients of Posterior Reversible Encephalopathy Syndrome with the clinical features: seizure activity in 97 (71%), consciousness impairment 39 (26%), headaches 39 (26%), visual impairment 39 (26%), acute hypertension 97 (67%), focal neurological deficit not reported. The radiological features in cases of bartynski et al were bilateral lesions in 98 out of 136 patient studied (72%), asymmetric lesions in 21 out of 136 patients (16%), grey matter involvement was not reported, posterior > anterior in 30 patients (22%), occipital in 34 patients (99%), parietal in 134 patients (99%), frontal in 93 patients (63%), temporal in 55 patients (40%). Some cases had brainstem, cerebellum and basal ganglion involvement.

Casey et al in 2000 reported the radiological features of Posterior Reversible Encephalopathy Syndrome in 16 patients of which bilateral lesion was seen in 11 (16) patients, posterior > anterior in 15 (94%), parietal 8, occipital, not reported surprisingly, frontal 15(88%), temporal 16(100%), brain stem and cerebellum – not reported, basal ganglia 3(19%).

McKinney et al in 2001 reported 76 cases of Posterior Reversible Encephalopathy Syndrome the most common clinical presentation he observed was seizure 76%, consciousness impairment 13% followed by headache and visual symptoms. The most common radiological feature in

his study was parietal – occipital (99%), followed by frontal (89%), temporal (68%). Few patients showed brainstem, cerebellum and basal ganglia involvement.

Lee in 2008 reported "36 cases of Posterior Reversible Encephalopathy Syndrome of which 20 were females, 16 were males with a mean age of presentation being 44.7 years. Hypertension (53%), renal disease (45%), malignancy (32%), transplantation (24%), dialysis (21%), were the co-morbid conditions noted. Clinical presentation was seizures (92%), visual symptoms (39%), and headache (53%). The mean systolic blood pressure recorded was 187 mmHg at presentation.

The clinical features resolved with treatment after about 5.3 days (mean average) the atypical features in MRI they reported were frontal lobe involvement in 22 (58%), grey matter 16 (42%), hemorrhage 2 (5%), unilateral lesion 2 (5%), infarct in 10 (26%), recurrent PRES 2 (5%), brainstem and cerebellum 22 (58%)".

Jennifer E fugate et al diagnosed 120 patients between 2005 to 2009 prospectively. The mean peak systolic blood pressure was 199 mm Hg. The mean peak diastolic blood pressure was 109 mmHg. Etiologies were hypertension 69 out of 120 patients (61%), cytotoxic drugs 21 (19%), preeclampsia or eclampsia 7(6%), sepsis 8 (7%), autoimmune

diseases 51 (45%), multiorgan dysfunction 1 (1%). The clinical features noted were seizure 84 (74%), altered sensorium 32 (28%), headache 29 (26%), visual symptoms 23 (20%). Imaging showed that parieto occipital regions were commonly involved 108 (94%) followed by frontal lobe involvement 88 (77%), temporal lobe 74 (64%) and cerebellar involvement in 63 (53%).

MATERIALS AND METHODS

Study Design : Descriptive study

Place of Study : Govt. Stanley Medical College and Hospital, Chennai-1.

Study Period : January 2012 to January 2014.

Study Population : All Patients with clinical suspicion of Posterior Reversible Encephalopathy Syndrome admitted/referred to the Neurology Department, Government Stanley Medical College and Hospital.

Inclusion Criteria:

Patients having clinical and radiological features consistent with Posterior Reversible Encephalopathy Syndrome

Exclusion Criteria:

Subcortical white matter lesions other than Posterior Reversible Encephalopathy Syndrome:

Infection – Progressive Multifocal Leukoencephalopathy.

Vascular – Posterior Circulation Stroke/ Superior Sagittal Sinus Thrombosis.

Neoplasms –Gliomatosis cerebri, Lymphoma, Glioma.

Demyelination- Acute disseminated encephalomyelitis, Multiple Sclerosis.

Dysmyelination- Leukodystrophies like Metachromatic leucodystrophy.

Metabolic- severe hypoglycemia, hypotension.

Methodology:

A descriptive study done by analyzing the medical records of patients diagnosed to have Posterior Reversible Encephalopathy Syndrome over a period of two years. The clinical data, etiological and radiological features of these patients were collected for the study purpose.

1) Consent to participate in the study was taken from the patients.
2) A descriptive study analyzing the patients diagnosed to have PRES over a period of 2 years was done.
3) Demographic data of the patients was obtained.
4) The clinical features with which the patients presented were carefully noted.

5) The MRI brain was done for all patients and the radiological features noted were recorded. The images were interpreted by the radiologists at Government Stanley medical college and Hospital.

6) The underlying cause or etiology of Posterior Reversible Encephalopathy Syndrome was identified and noted in each case.

7) The treatment given and the clinical outcome after treatment were noted.

8) Persistent neurological deficit if any was recorded.

Statistical Analysis

Statistical analysis was performed using SPSS (Statistical Package for Social Sciences) 16.0 software. The data collected as per the proforma was tabulated, using the above software analysis was done and results were obtained.

RESULTS

1. There was a striking female preponderance in patients with Posterior Reversible Encephalopathy Syndrome, in this study, with 27 (87.1 %) of females and 4 (12.9 %) of males.

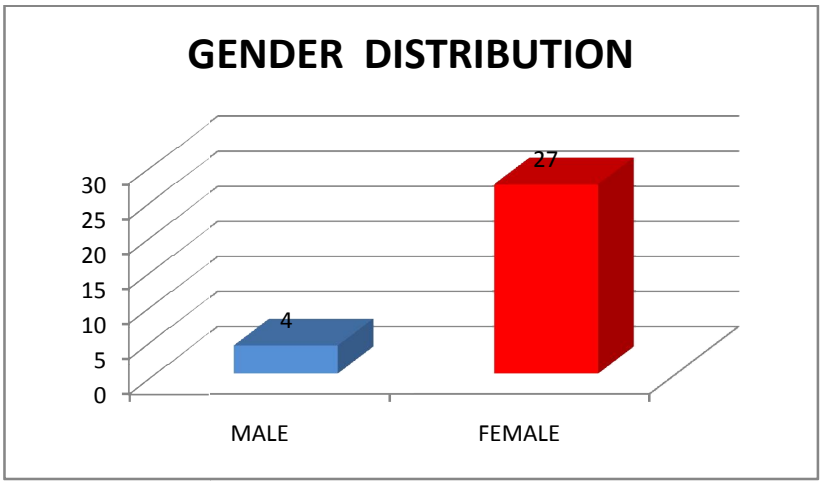


Fig 1: Gender Distribution of patients with PRES

2. The age of patients ranged from 15 years to 65 years. The mean age being 26.34 years. The common age group of Posterior Reversible Encephalopathy Syndrome in this study was 20 to 39 years, 64% of the patients were in this age group.

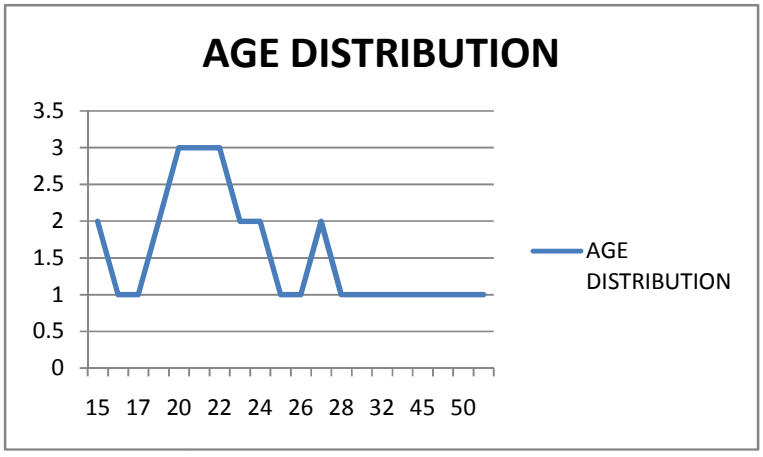


Fig 2: Age Distribution of PRES

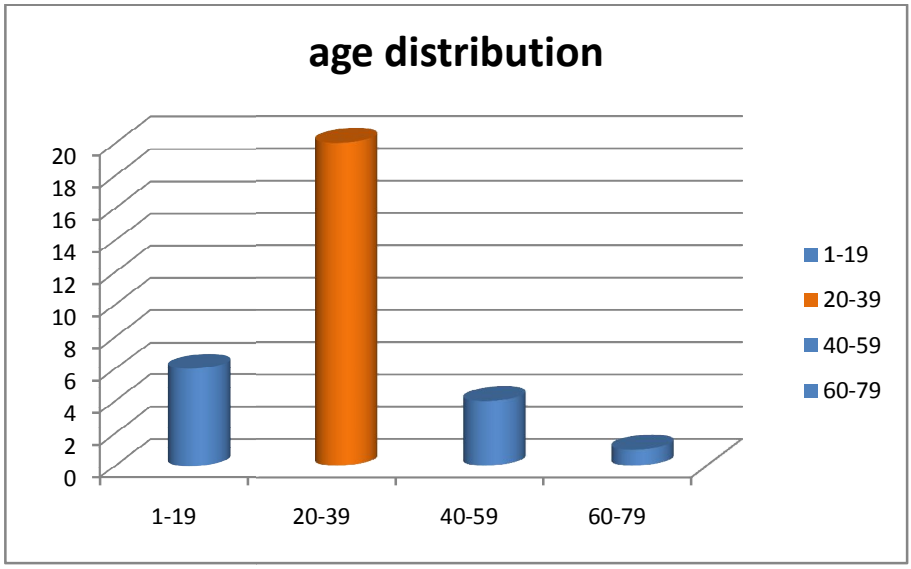


Fig 3: Age Distribution in PRES showing that most of the patients affected are in the age group between 20 – 39 years.

3. The duration of symptoms before presentation to the hospital was a minimum of few hours to one day to a maximum of two weeks.

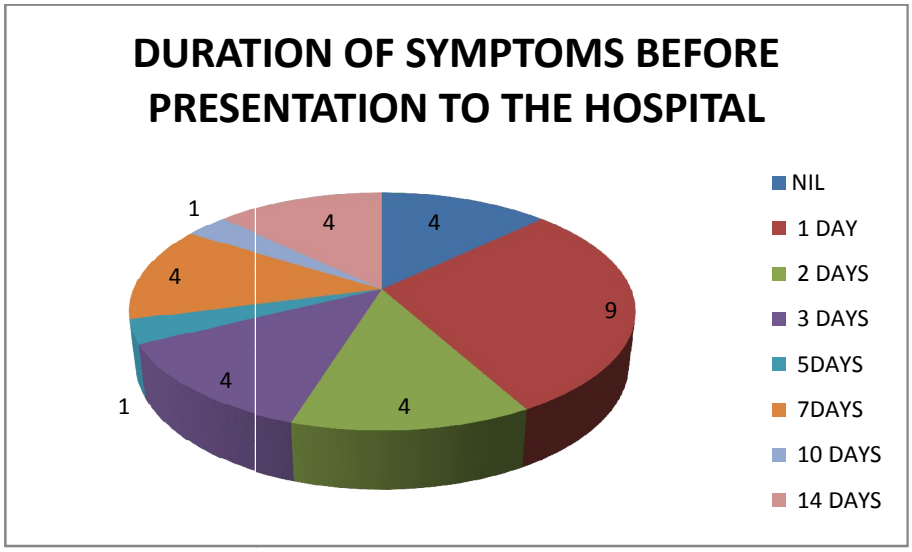


Fig 4: Diagram showing duration of the symptoms before presentation to the hospital

4. The most common presenting complaint of Posterior Reversible Encephalopathy Syndrome in this study was headache. 87.09% of the patients had headache as the presenting complaint in this study.

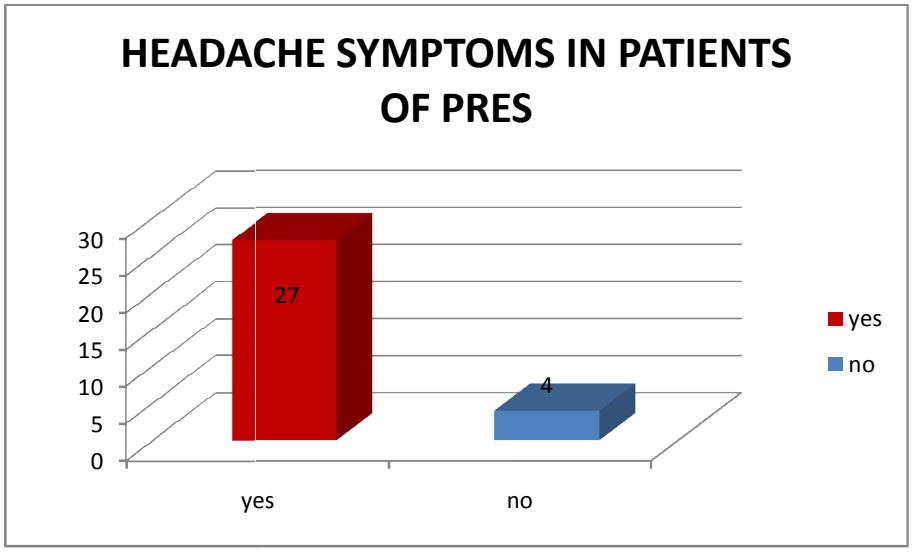


Fig 5: Incidence of Headache in PRES

5. In this study, seizures were the second most common symptom of PRES. Seizures were seen in 64.51% of the patients in this study. The frequency of seizures ranged from one to several seizures a day.4 of our patients, in this study presented with status epilepticus.

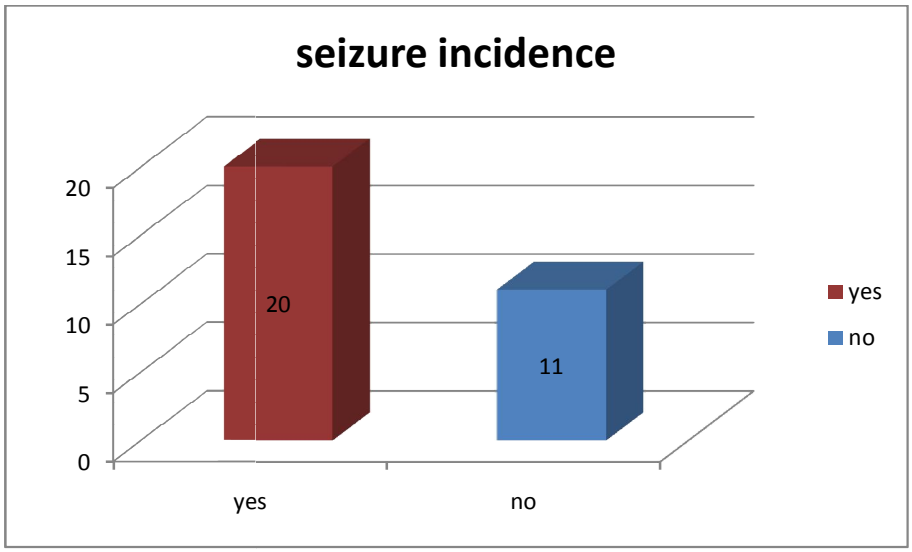


Fig 6: Seizures in Patients with PRES

6. Visual symptoms were seen in 48.38% of the patients.

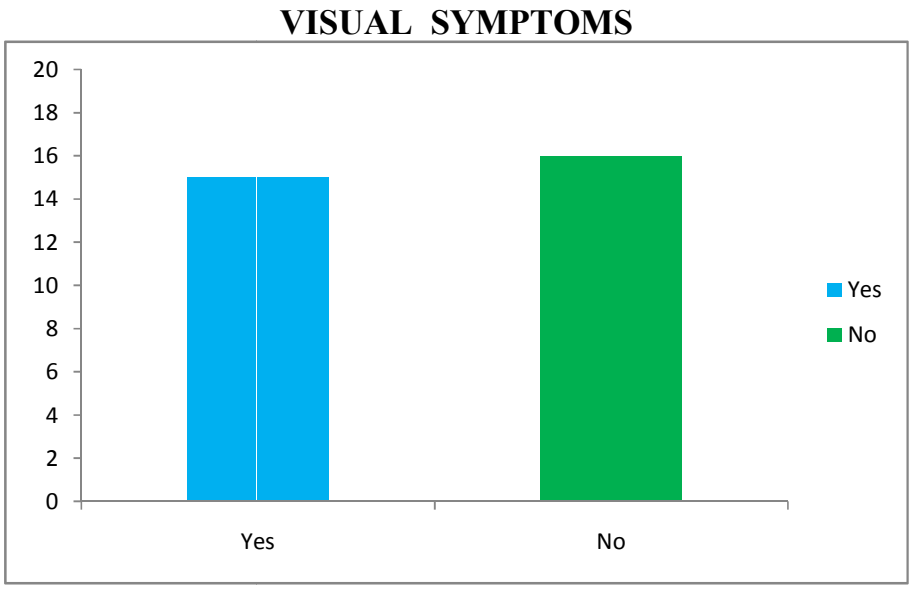


Fig 7: Visual Symptoms in Patients of PRES

7. Impaired consciousness was noted in 51.61% of the patients of Posterior Reversible Encephalopathy Syndrome in this study

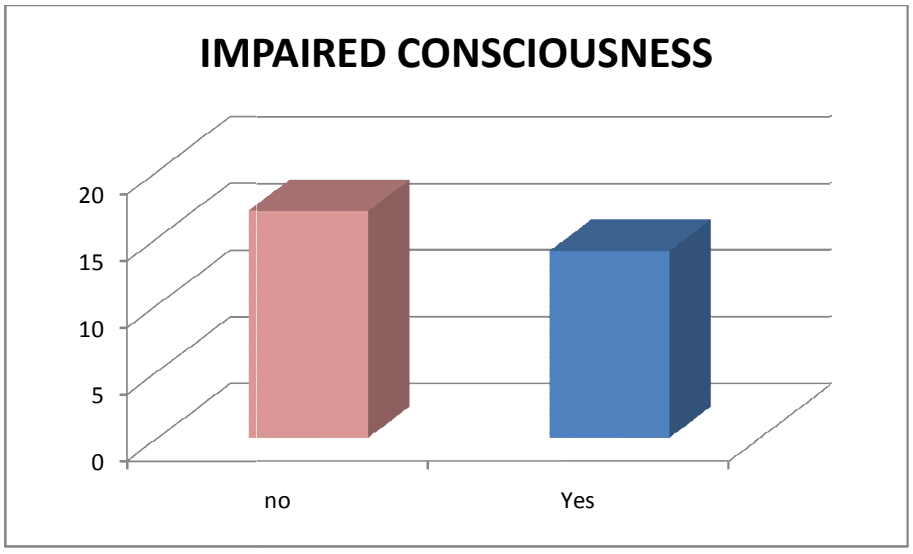


Fig 8: Impaired consciousness in patients with PRES

8. 15 out of the 31 patients in this study had visual complaints. The common visual complaints seen in the patients of PRES in this study were flashes of light, blurring of vision, flickers of light to field defects and cortical blindness. 48.38% of the patients in this study presented with visual symptoms. This shows almost 50% of patients presented with visual symptoms.

9. Many patients had various combination of all symptoms, sometimes in isolation as well, like headache alone, in 1 patient with pre eclampsia and impaired consciousness alone, in a patient of Posterior Reversible Encephalopathy Syndrome with ESRD (End Stage Renal Disease) on hemodialysis.

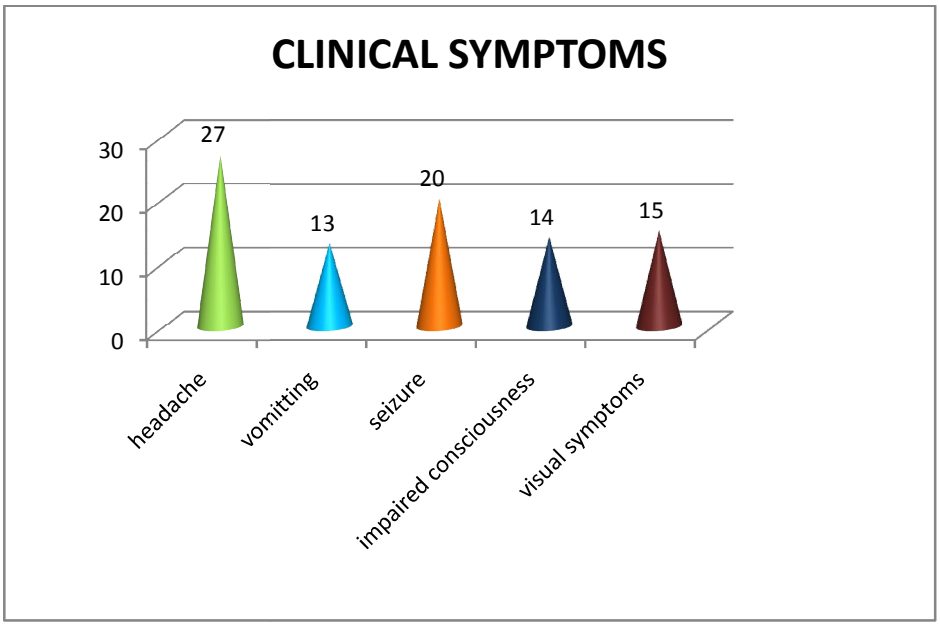


Fig 9: Presenting symptoms of PRES – headache and seizures were the commonest presenting symptoms in this study

10. There was a varied aetiology of Posterior Reversible Encephalopathy Syndrome in this study. 18 out of the 31 cases i.e. 58.06% of cases were pregnancy related PRES in this study.

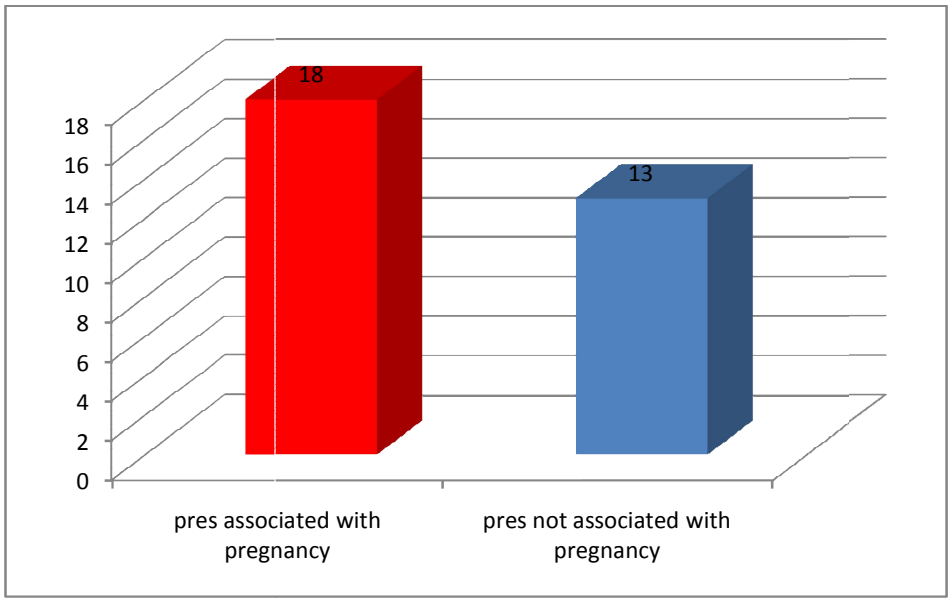


Fig 10: Distribution of Pregnancy related PRES vs. Non Pregnancy related PRES.

11. The other causes, non pregnancy associated cases were primary hypertension in 6 of the 31 cases (16.12% of patients). Others were SLE, PIGN, PAN, ESRD on hemodialysis, OPC poisoning with toxic neuropathy and dysautonomia, porphyria with SIADH and systemic hypertension and Takayasu`s arteritis. All the above aetiologies were present in 1 case each (3.225% each) out of the 31 patients.

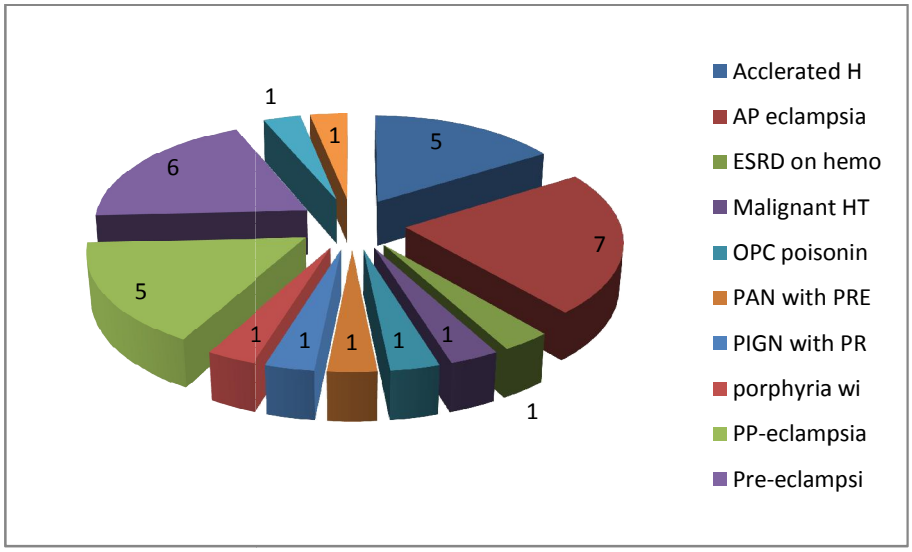


Fig 11: Distribution of etiologies of PRES in this study

12. The duration of headache, at the time of first presentation to the hospital, ranged from 1 day to 14 days.

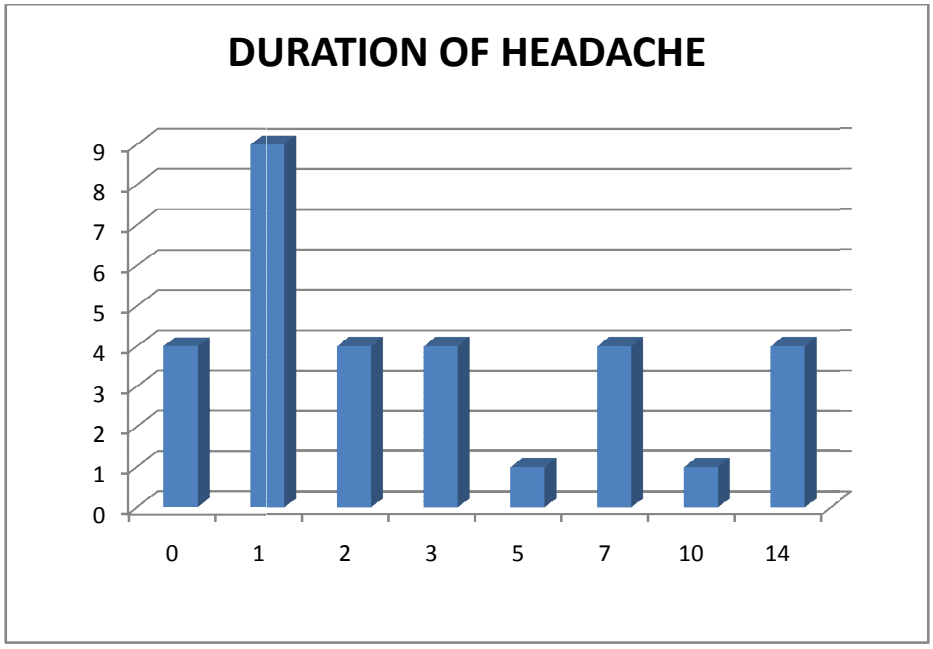


Fig 12: Duration of headache at the time of presentation to hospital.

Vertical column : no of patients

Horizontal column : no of days of headache.

13. Amongst the pregnancy associated Posterior Reversible Encephalopathy Syndrome, pre-eclampsia was seen in 6 cases (19.35 %), antepartum eclampsia was seen in 8 cases (25.80%) and postpartum eclampsia in 4 cases (12.90%), out of the total of 31 patients.

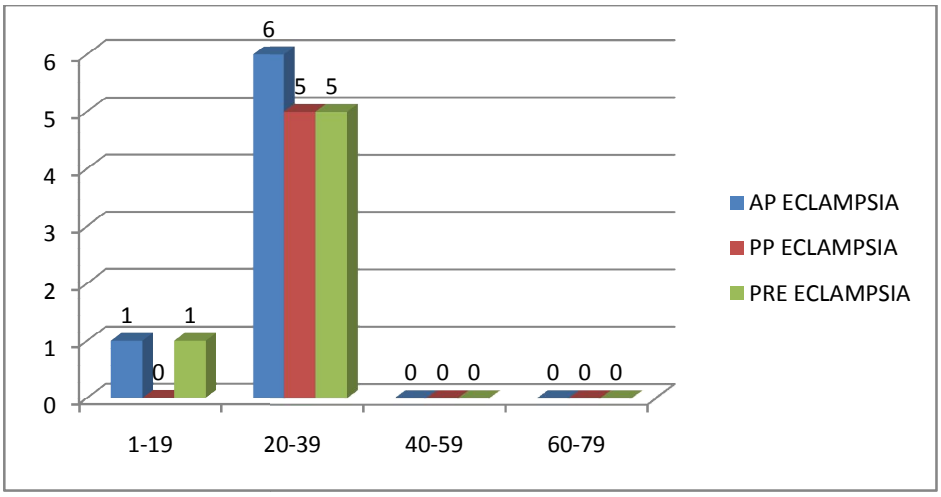


Fig 13: Pregnancy associated PRES

14. Hypertension was detected in 21 out of 31 patients. Amongst them, 8 patients had pregnancy associated hypertension and 13 had non pregnancy associated hypertension.

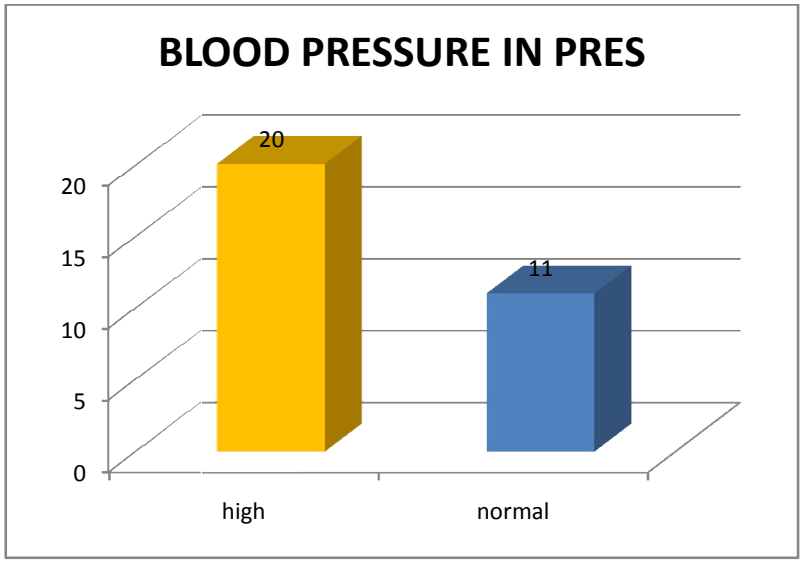


Fig 14: Blood pressure in PRES

15. 11 of the 31 patients i.e. 32.25 % of patients had normal blood pressure. This supports the fact that the abrupt raise in blood pressure above the baseline is more important than the absolute value of blood pressure in causing Posterior Reversible Encephalopathy Syndrome

16. Amongst the 13 non pregnancy associated hypertension 2 had transient hypertension- 1 following PIGN, 1 following dysautonomia of Gullian Barre syndrome. Fluctuating hypertension was found in a patient with porphyria secondary to dysautonomia. Persistent hypertension was seen in 6 patients with primary hypertension, 1 patient with PAN, 1 patient with Takayasu's arteritis. 1 patient with ESRD and 1 patient with blunt injury abdomen.

17. The patient with SLE though had no documented hypertension it was postulated that probably she had transient hypertension that was undocumented following the reversible nephritis she had.

18. The patient with blunt injury abdomen following RTA had persistent hypertension, though a definite cause for hypertension

was not deducted - this patient had injury, contusion to the lower pole of right kidney. It was postulated probably he had renovascular injury as well that would have caused the hypertension and Posterior Reversible Encephalopathy Syndrome. However his renal artery Doppler study was normal.

19. Amongst the 31 patients on examination 7 patients (22.58%) had papillodema. All these patients had documented hypertension, 3 had PIH, and 4 had accelerated hypertension secondary to primary hypertension.

20. Radiological features in these patients were, occipital lobe involvement was seen in 24 out of 31 patients i.e.77.41 % of patients had occipital lobe involvement, parietal lobe involvement was seen in 21 out of 31 patients (67.74 %), both occipital and parietal lobe involvement was seen in 16 out of the 31 patients (51.61 %). Temporal lobe involvement was seen in 15 out of 31 (48.38 %). Frontal lobe involvement was seen in 6 out of 31 patients (19.35 %), other area involvement was seen in 2 out of 31 patients (6.45%) – 1 patient with postpartum eclampsia had bilateral capsuloganglionic vasogenic edema with no diffusion

restriction, one another patient with primary hypertension had bilateral capsuloganglionic, bilateral corona radiata, bilateral centrum semiovale, brainstem and cerebellar vasogenic edema without diffusion restriction.

21. In this study the commonest lobe involved was occipital (77.4%) followed by parietal (67.74%), followed by temporal (48.38%), further followed by frontal (19.35%) and other areas including brain stem (6.45%).

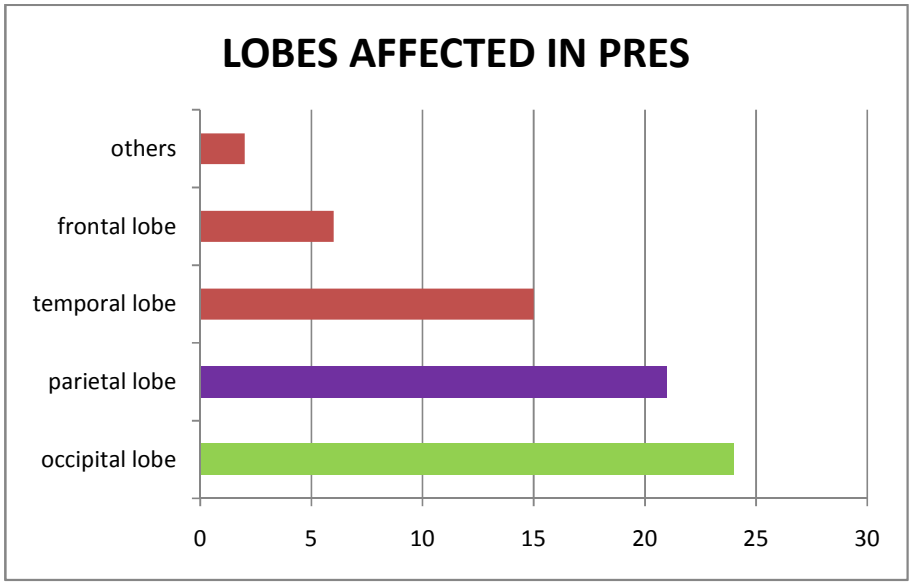


Fig 15: Lobes affected in PRES

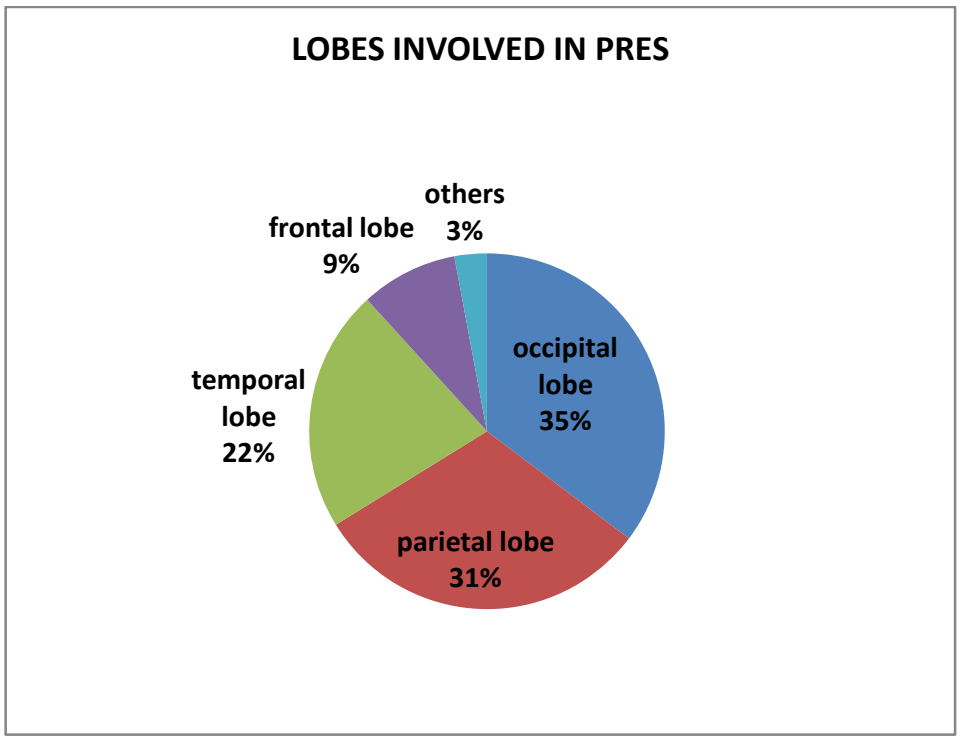


Fig 16: Lobes involved in PRES

1. Symmetrical lesions were found in 21 out of the 31 patients (67.74%). 10 patients had asymmetrical lesions (32.25%).

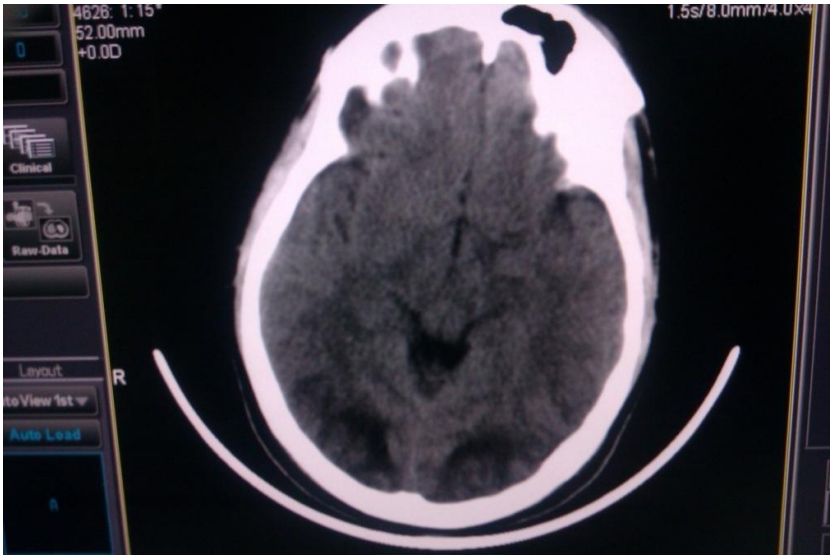


Fig 17: CT Brain showing symmetrical hypodensity in the bilateral occipital lobes in a case of PRES

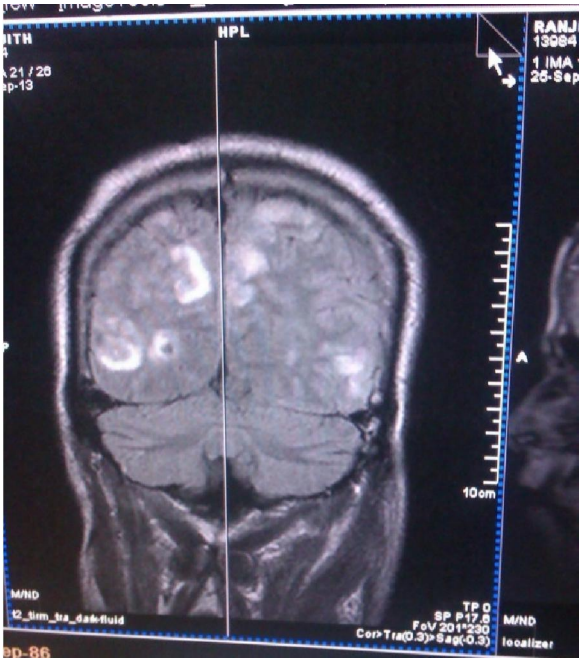


Fig 18

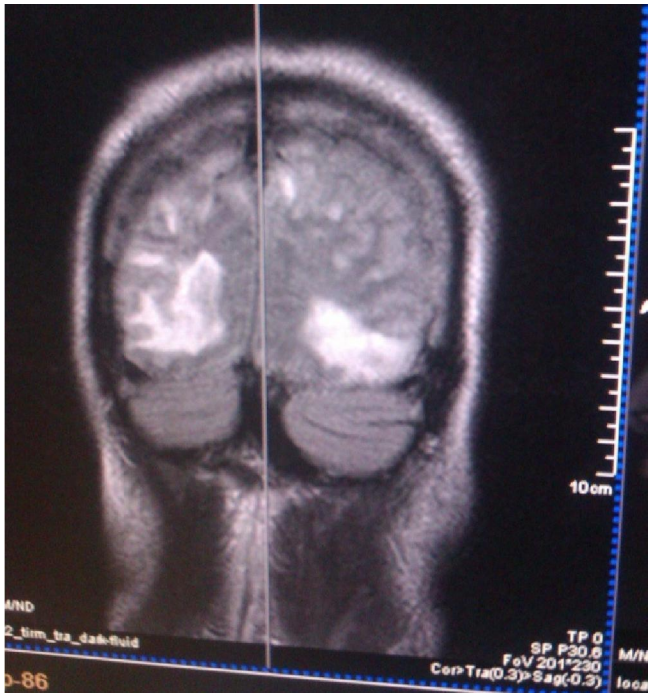


Fig 18 & 19: MRI Brain T2 FLAIR coronal showing bilateral parieto-occipital hyperintensities in a case of PRES

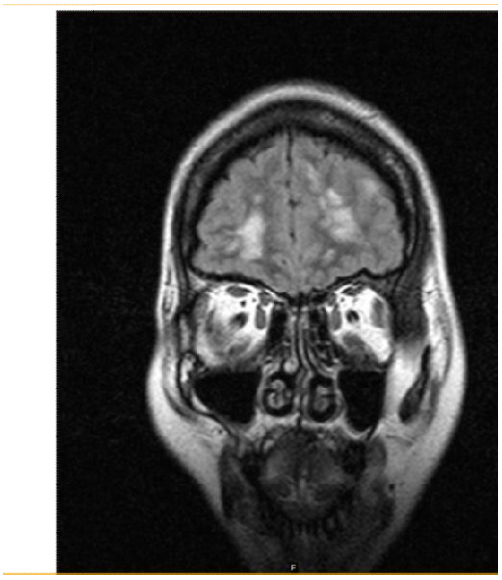


Fig 20: MRI Brain T2 FLAIR coronal showing subcortical white matter hyperintensities in the bilateral frontal lobes

2. All the patients had vasogenic edema, the hallmark of PRES, commonly seen in the subcortical regions of the bilateral parieto-occipital lobes.

VASOGENIC EDEMA-PATHOGNOMONIC OF PRES

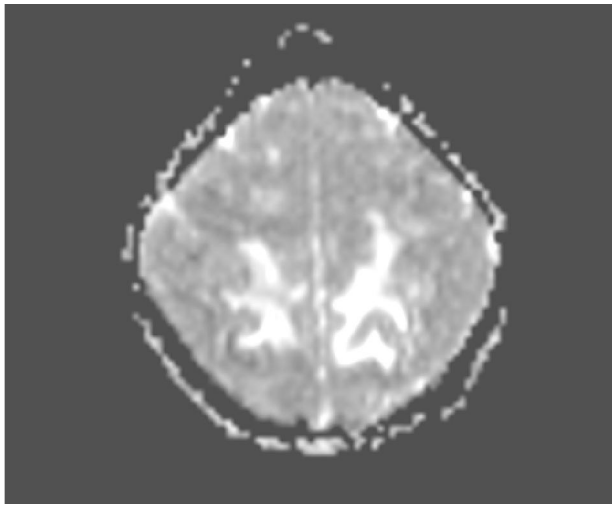


Fig 21: MRI ADC sequence: showing a hyperintense area in the posterior subcortical region of brain.

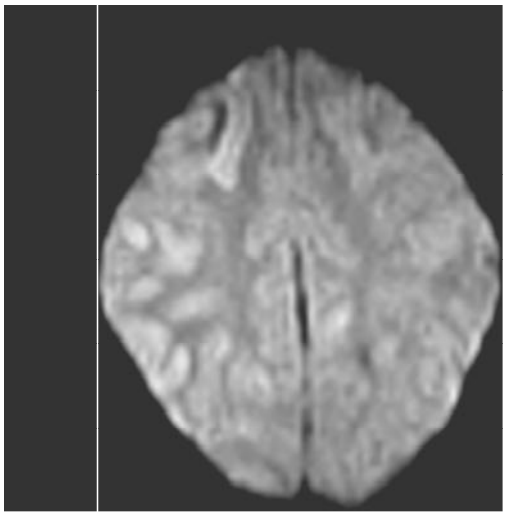


Fig 22: MRI Brain DWI sequence : showing isointense area in the corresponding posterior subcortical region suggestive of vasogenic edema.

3. Subcortical lesions were found in all the 31 patients. Cortical lesion was seen in 14 out of the 31 patients (45.16%). 15 patients i.e. 48.38% of the patients had both cortical and subcortical lesions.

4. Diffusion restriction was noted in 4 out of 31 patients i.e. 12.90% of the patients showed diffusion restriction.

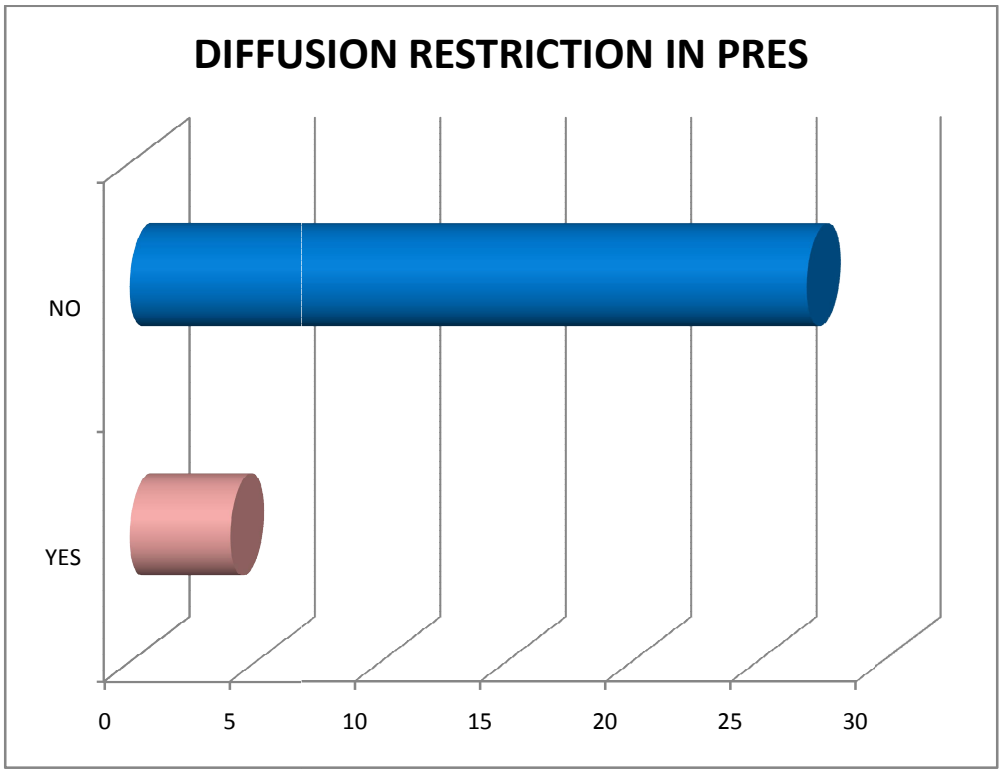


Fig 21: Diffusion Restriction – complication of PRES

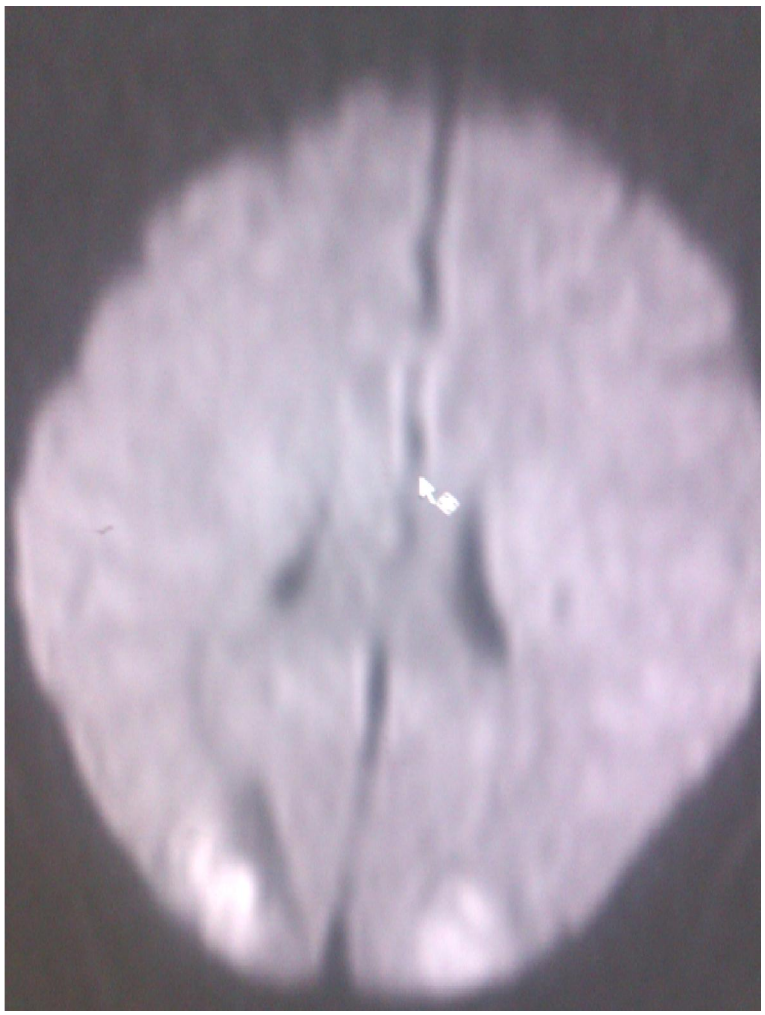


Fig 22: DWI Brain showing diffusion bright lesion in the bilateral occipital regions –complication of PRES – Infarct showing Diffusion Restriction

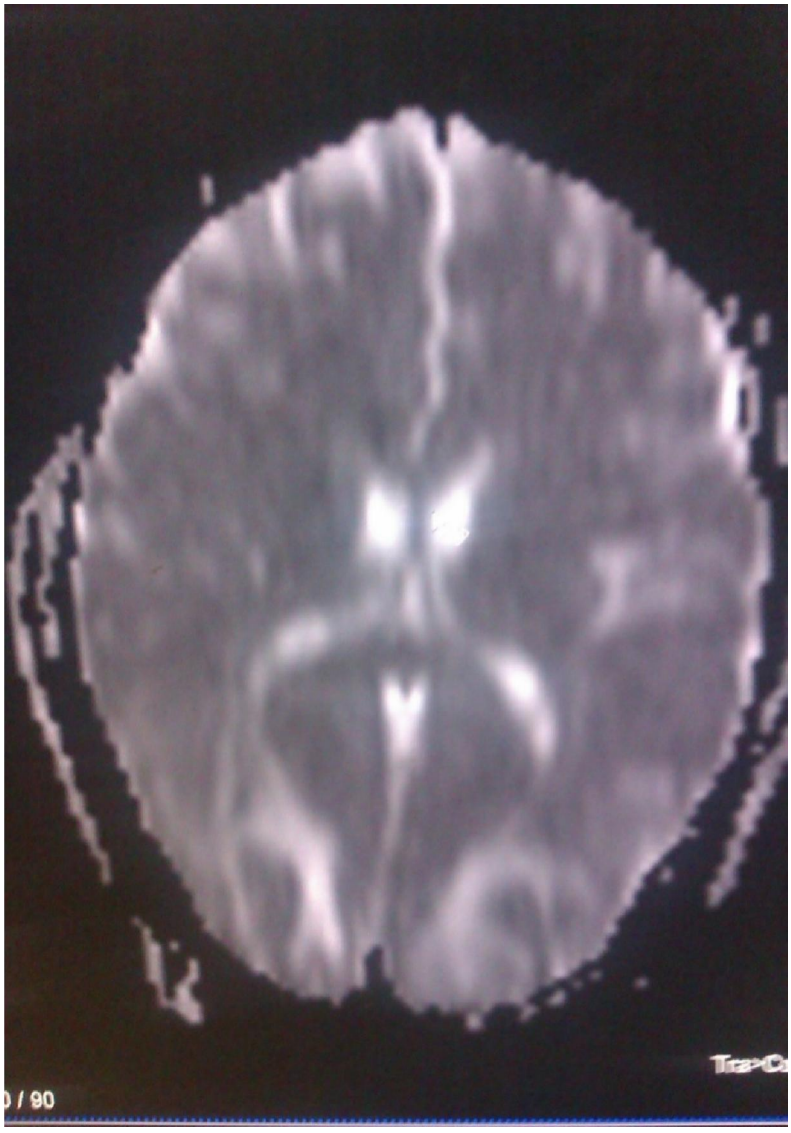
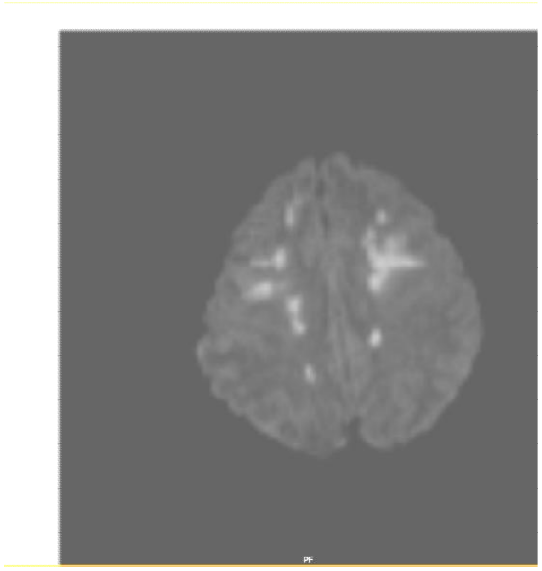


Fig 23: ADC Brain showing hypointense lesions in the corresponding bilateral occipital region-complication of PRES - Infarct.



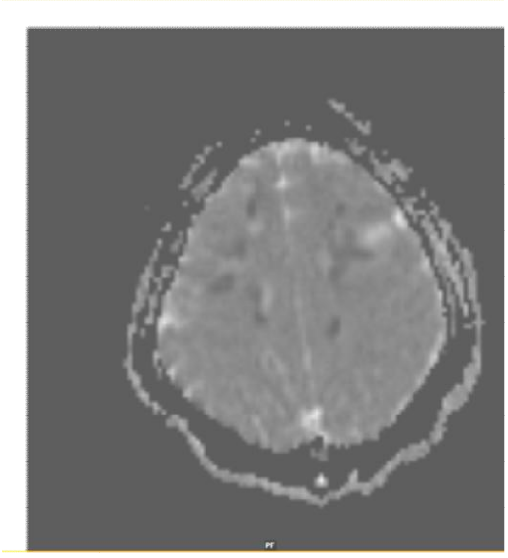


Fig 24 & 25: showing diffusion restriction in the bilateral parietal regions

5. Though Posterior Reversible Encephalopathy Syndrome is largely reversible, sometimes there can be irreversible complications. In this study 28 patients had reversible complications, 2 patients had irreversible (visual defects) complications and 1 patient died.

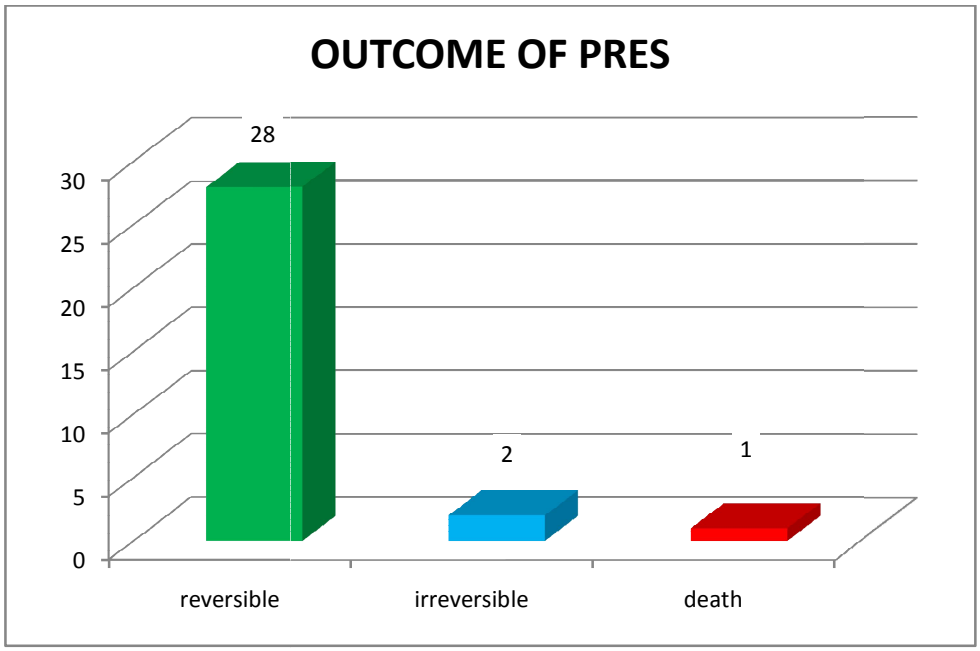


Fig 25: Outcome of PRES

6. Recurrent PRES can occur rarely. One of the patient with ESRD on dialysis developed PRES twice, at an interval of 6 months, following renal hypertension.

SUMMARY OF THE RESULTS

1. Females are predominantly affected with F: M ratio = 7:1.
2. The commonest age group affected in this study is between 22 – 39 years.
3. Headache is the commonest symptom of Posterior Reversible Encephalopathy Syndrome in this study.
4. Duration of headache ranged from ≤ 1 day up to 14 days, at the time of presentation to the hospital.
5. The commonest aetiology of Posterior Reversible Encephalopathy Syndrome is pregnancy induced hypertension. 18 patients (58.06%) out of the 31 cases in this study were pregnancy related. All these patients were primigravida except one. Amongst the 18 patients 8 had antepartum eclampsia, 6 had preeclampsia and 4 had postpartum eclampsia.
6. Hypertension was documented in 21(67.74%) out of the 31 patients. The range of hypertension varied from 130/96 to as high as 210/140 mmHg.
7. With the control of blood pressure symptoms resolved promptly. One of the patient with bilateral cortical blindness regained normal vision within 4 hours after blood pressure control.
8. On examination papillodema was seen in 7 patients (22.58%) of the patients out of the 31 cases.
9. The commonest lobes involved were the occipital 77.41% and parietal 67.74%.

10. Vasogenic edema, the prime radiological finding of Posterior Reversible Encephalopathy Syndrome was seen in all the patients in this study.
11. Symmetrical lesion on imaging was seen in 67.74% of the patients.
12. All had subcortical white matter changes. 48.38% of the patients had both subcortical and cortical involvement
13. Diffusion restriction was seen in 12.9% of the patients.
14. In most of the cases the symptoms were reversible. However irreversible complication (visual defects) was seen in 2 (6%) of the patients. 1 patient died due to worsening of the primary illness.
15. Recurrent PRES was seen in 1 out of the 31 (3.22%) of the patients.

DISCUSSION

Posterior Reversible Encephalopathy Syndrome is common in females as most of the cases are pregnancy related. In this study also there was a marked female preponderance with 87% of the patients being females and only 13% being males.

Posterior Reversible Encephalopathy Syndrome commonly affects young and middle age individuals, the mean age being 39 years to 47years in various studies (14).

In this study Posterior Reversible Encephalopathy Syndrome was common in the ages 20 – 39 years with the mean of 26.34 years. This finding is consistent with the feature that this disease entity is common in the young and middle age group (1)(14).

Also in a case study from India – case series of 20 patients by Praveen Kumar et al, with Posterior Reversible Encephalopathy Syndrome 60 % of the patients were in the age group 20 – 30 years, a finding almost similar to our study.

Posterior Reversible Encephalopathy Syndrome has been reported in patients who are 4 years old up to 90 years old(14). Hinchey reported that the youngest patient in their case series was 15 years and the oldest

was 62 years. In our case study the youngest was 15 years of age and the oldest was 65 years of age.

In most of the studies worldwide, impaired consciousness was the commonest symptom of Posterior Reversible Encephalopathy Syndrome (1,2,3,7,19). However in our study headache (87.59%) and seizures (64.51%) were the commonest symptoms of Posterior Reversible Encephalopathy Syndrome.

Praveen Kumar et al reported headache (75%) as the most common symptom of Posterior Reversible Encephalopathy Syndrome followed by seizures, as seen in our study. Abrupt raise in blood pressure of any etiology (as encountered in pregnancy related hypertension) is often the cause of Posterior Reversible Encephalopathy Syndrome.

Pregnancy related Posterior Reversible Encephalopathy Syndrome was the commonest cause of Posterior Reversible Encephalopathy Syndrome in our study followed by primary hypertension. Praveen Kumar et al also reported that pregnancy induced hypertension (55%) as the commonest etiology of Posterior Reversible Encephalopathy Syndrome followed by primary hypertension. This was consistent with Petrovic et al, Lee et al, Casey et al studies that primary hypertension and pregnancy induced hypertension are the commonest etiology. Also in

Jennifer Fugate et al study Primary Hypertension was the commonest cause. However Hinchey et al, Legriel et al, found immunosuppressive drugs following transplantation was the commonest cause.

Hinchey et al, Burnett et al, Bartynski et al documented hypertension in 67 – 80 % of the patients; in our study hypertension was noted in 67.74% of the patients.

In the radiological findings, occipital lobe involvement was seen in 93 %(1) to 99 %(3, 19) of the patients followed by parietal 87%(1), 99 %(3), 67%(12) 50%(6). In our study also the commonest lobes affected were occipital 77.41% and parietal 67.74% lobes.

Symmetrical lesions were reported in 100%(7) 69%(6) of the patients. In our study the symmetrical pattern was seen in 67.74% of the patients.

Asymmetrical pattern was seen in 32.25% of the patients. Subcortical involvement was seen in all the patients. Cortical involvement was seen in 13 out of the 31 patients i.e. 41.93% of the patients had cortical involvement. Also this means that the same 41.93% of the patients had both subcortical and cortical involvement.

Vasogenic edema (iso or hypointense in DWI and hyperintense in ADC) pathognomonic of Posterior Reversible Encephalopathy Syndrome was seen in all cases especially in the posterior subcortical regions.

Rarely there can be recurrent Posterior Reversible Encephalopathy Syndrome. Recurrent Posterior Reversible Encephalopathy Syndrome had been reported in 6% of the case(12).In our study one patient (3.22%) had recurrent Posterior Reversible Encephalopathy Syndrome. Legriel et el reports 15% mortality rate in Posterior Reversible Encephalopathy Syndrome.

Lee et al (14) reported persistent neurological deficit was seen in 26% of the patients. In our study two of the patients had irreversible complications (visual defects) and one patient died of the primary illness. The mortality rate in our study was 3.22%.

CONCLUSION

* Posterior Reversible Encephalopathy Syndrome is common in females, with a female : male ratio of 7 :1.

* The commonest age groups affected are the young and the middle aged, 64%of the patients were in this age group.

* Headache (87.09%) is the commonest symptom of Posterior Reversible Encephalopathy Syndrome in this study. Seizures (64.51%) was the second most common symptom in this study. One half of the patients had the other symptoms of PRES - impaired consciousness, visual symptoms and vomiting.

* Papillodema is seen in nearly 1/3rd of the patients.

* Not all patients have documented hypertension. Hypertension was documented in 64.75% of the patients in this study, the rest, 32.26% of the patients had no hypertension signifying that the abrupt rise of blood pressure from the baseline, is more important

than the absolute value of blood pressure in causing Posterior Reversible Encephalopathy Syndrome.

* The commonest cause is Pregnancy related Posterior Reversible Encephalopathy Syndrome, with almost all the pregnant patients being primigravida.

* There is varied aetiology for Posterior Reversible Encephalopathy Syndrome, ranging from the commoner pregnancy related Posterior Reversible Encephalopathy Syndrome to the rare causes like porphyria, Posterior Reversible Encephalopathy Syndrome following blunt injury abdomen, following OPC poisoning etc, as seen in this study. Hence a high index of suspicion and a thorough knowledge of conditions that could predispose to Posterior Reversible Encephalopathy Syndrome is essential for the early diagnosis.

* The commonest radiological pattern is subcortical, symmetrical vasogenic edema in the occipoparietal regions. However cortical involvement was seen in nearly 50% of the patients. Asymmetrical involvement was seen in nearly 1/3rd of the patients.

* Recurrent PRES is observed in a small percentage (3.22%) of patients.
* Prompt control of blood pressure prevents irreversible complications.
* A high index of clinical suspicion in appropriate settings along with radiological correlation, will help in early recognition and prompt management of PRES, which will aid in preventing grave complications.

BIBLIOGRAPHY

1. Hinchey J, Chaves C, Appignani B, et al (1996) A reversible posterior leukoencephalopathy syndrome. N Engl J Med 334: 494–500

2. Bartynski WS (2008) Posterior reversible encephalopathy syndrome, part 1: fundamental imaging and clinical features. AJNR Am J Neuroradiol 29: 1036–1042

3. Bartynski WS (2008) Posterior reversible encephalopathy syndrome, part 2: controversies surrounding Pathophysiology of vasogenic edema. AJNR Am J Neuroradiol 29: 1043–1049

4. Schwartz RB, Jones KM, Kalina P, et al (1992) Hypertensive encephalopathy: findings on CT, MR imaging, and SPECT imaging in 14 cases. AJR Am J Roentgenol 159: 379–383

5. Schwartz RB, Bravo SM, Klufas RA, et al (1995) Cyclosporine neurotoxicity and its relationship to hypertensive encephalopathy: CT and MR findings in 16 cases. AJR Am J Roentgenol 165: 627–631

6. Casey SO, Sampaio RC, Michel E, Truwit CL (2000) Posterior reversible encephalopathy syndrome: utility of fluid-attenuated inversion recovery MR imaging in the detection of cortical and subcortical lesions. AJNR Am J Neuroradiol 21: 1199–1206

7. Lee VH, Wijdicks EF, Manno EM, Rabinstein AA (2008) Clinical spectrum of reversible posterior leukoencephalopathy syndrome. Arch Neurol 65: 205–210

8. Garg RK Post grad Med J. 2001 Jan;77(903):24-8. Posterior leukoencephalopathy syndrome.

9. Rodrigo Pedraza, Paul E. Marik, Joseph Varon. Crit Care & Shock (2009) 12:135-143. Posterior Reversible Encephalopathy Syndrome: A Review

10. Posterior Reversible Encephalopathy Syndrome: Associated Clinical and Radiologic Findings Jennifer E.Fugate, DO, Daniel O, Classen, MD, Harry J.Cloft, MD, PhD, Mayo Clin Proc; 2010 May; 85(5): 427-432

11. Dillon WP, Rowley H. Am J Neuroradiol 1998; 19:591 The Reversible Posterior Cerebral Edema Syndrome

12. Burnett MM, Hess CP, Roberts JP, Bass NM, Douglas VC, Josephson SA (2010) Presentation of reversible posterior leukoencephalopathy syndrome in patients on calcineurin inhibitors. Clin Neurol Neurosurg 112: 886–889

13. Servillo G, Striano P, Striano S, et al (2003) Posterior reversible encephalopathy syndrome (PRES) in critically ill obstetric patients. Intensive Care Med 29: 2323–2326

14. Legriel,pico, azoulay – understanding posterior reversible encephalopathy syndrome. Annual update in intensive care and emergency medicine 2011

15. Kozak OS, Wijdicks EF, Manno EM, Miley JT, Rabinstein AA (2007) Status epilepticus as initial manifestation of posterior reversible encephalopathy syndrome. Neurology 69: 894–897

16. Bartynski WS, Boardman JF (2007) Distinct imaging patterns and lesion distribution in posterior reversible encephalopathy syndrome. AJNR Am J Neuroradiol 28: 1320–1327

17. Bartynski WS, Boardman JF, Zeigler ZR, Shadduck RK, Lister J (2006) Posterior reversible encephalopathy syndrome in infection, sepsis, and shock. AJNR Am J Neuroradiol 27: 2179–2190

18. Covarrubias DJ, Luetmer PH, Campeau NG (2002) Posterior reversible encephalopathy syndrome: prognostic utility of quantitative diffusion-weighted MR images. AJNR Am J Neuroradiol 23: 1038–1048

19. McKinney AM, Short J, Truwit CL, et al (2007) Posterior reversible encephalopathy syndrome: incidence of atypical regions of involvement and imaging findings. AJR Am J Roentgenol 189: 904–912

20. Ducros A, Boukobza M, Porcher R, Sarov M, Valade D, Bousser MG (2007) The clinical and radiological spectrum of reversible cerebral vasoconstriction syndrome. A prospective series of 67 patients. Brain 130: 3091–3101

21. Hefzy HM, Bartynski WS, Boardman JF, Lacomis D (2009) Hemorrhage in posterior reversible encephalopathy syndrome: imaging and clinical features. AJNR Am J Neuroradiol 30: 1371–1379

22. Belogolovkin V, Levine SR, Fields MC, Stone JL (2006) Postpartum eclampsia complicated by reversible cerebral herniation. Obstet Gynecol 107: 442–445

23. Lamy C, Mas JL (2001) [Reversible posterior leukoencephalopathy. A new syndrome or a new name for an old syndrome?]. Presse Med 30: 915–920

24. Legriel S, Bruneel F, Spreux-Varoquaux O, et al (2008) Lysergic acid amide-induced posterior reversible encephalopathy syndrome with status epilepticus. Neurocrit Care 9: 247–252

25. Cole AJ (2004) Status epilepticus and periictal imaging. Epilepsia 45(Suppl 4): 72–77

26. Thurnher MM, Post MJ, Rieger A, Kleibl-Popov C, Loewe C, Schindler E (2001) Initial and follow-up MR imaging findings in AIDS-related progressive multifocal leukoencephalopathy treated with highly active antiretroviral therapy. AJNR Am J Neuroradiol 22: 977–984

27. O'Sullivan M (2008) Leukoaraiosis. Pract Neurol 8: 26–38

28. Chabriat H, Joutel A, Dichgans M, Tournier-Lasserve E, Bousser MG (2009) Cadasil. Lancet Neurol 8: 643–653

29. Gilman S (1998) Imaging the brain. First of two parts. N Engl J Med 338: 812–820

30. Sonneville R, Klein IF, Wolff M (2010) Update on investigation and management of postinfectious encephalitis. Curr Opin Neurol 23: 300–304

31. Matthews PM, Tampieri D, Berkovic SF, et al (1991) Magnetic resonance imaging shows specific abnormalities in the MELAS syndrome. Neurology 41: 1043–1046

32. Pomper MG, Miller TJ, Stone JH, Tidmore WC, Hellmann DB (1999) CNS vasculitis in autoimmune disease: MR imaging findings and correlation with angiography. AJNR Am J Neuroradiol 20: 75–85

33. Kallenberg K, Schulz-Schaeffer WJ, Jastrow U, et al (2006) Creutzfeldt-Jakob disease: comparative analysis of MR imaging sequences. AJNR Am J Neuroradiol 27: 1459–1462

34. Stam J (2005) Thrombosis of the cerebral veins and sinuses. N Engl J Med 352: 1791–1798

35. Culebras A, Kase CS, Masdeu JC, et al (1997) Practice guidelines for the use of imaging in transient ischemic attacks and acute stroke. A report of the Stroke Council, American Heart Association. Stroke 28: 1480–1497

36. Vaughan CJ, Delanty N (2000) Hypertensive emergencies. Lancet 356: 411–417

37. Baethmann A, Maier-Hauff K, Kempski O, Unterberg A, Wahl M, Schurer L (1988) Mediators of brain edema and secondary brain damage. Crit Care Med 16: 972–978

38. Ferrara JL (2000) Pathogenesis of acute graft-versus-host disease: cytokines and cellular effectors. J Hematother Stem Cell Res 9: 299–306

39. Antin JH, Ferrara JL (1992) Cytokine dysregulation and acute graft-versus-host disease. Blood 80: 2964–2968

40. Holler E, Kolb HJ, Moller A, et al (1990) Increased serum levels of tumor necrosis factor alpha precede major complications of bone marrow transplantation. Blood 75: 1011–1016

41. Schots R, Kaufman L, Van Riet I, et al (2003) Proinflammatory cytokines and their role in the development of major transplant-related complications in the early phase after allogeneic bone marrow transplantation. Leukemia 17: 1150–1156

42. de Vries HE, Kuiper J, de Boer AG, Van Berkel TJ, Breimer DD (1997) The blood-brain barrier in neuroinflammatory diseases. Pharmacol Rev 49: 143–155

43. Stanimirovic D, Satoh K (2000) Inflammatory mediators of cerebral endothelium: a role in ischemic brain inflammation. Brain Pathol 10: 113–126

44. Narushima I, Kita T, Kubo K, et al (2003) Highly enhanced permeability of blood-brain barrier induced by repeated administration of endothelin-1 in dogs and rats. Pharmacol Toxicol 92: 21–26

45. Wijdicks EF (2001) Neurotoxicity of immunosuppressive drugs. Liver Transpl 7: 937–942

46. Bartynski WS, Boardman JF (2008) Catheter angiography, MR angiography, and MR perfusion in posterior reversible encephalopathy syndrome. AJNR Am J Neuroradiol 29: 447–455

47. Brubaker LM, Smith JK, Lee YZ, Lin W, Castillo M (2005) Hemodynamic and permeability changes in posterior reversible encephalopathy syndrome measured by dynamic susceptibility perfusion-weighted MR imaging. AJNR Am J Neuroradiol 26: 825–830

48. Engelter ST, Petrella JR, Alberts MJ, Provenzale JM (1999) Assessment of cerebral microcirculation in a patient with hypertensive encephalopathy using MR perfusion imaging. AJR Am J Roentgenol 173: 1491–1493

49. Wang X, Lo EH (2003) Triggers and mediators of hemorrhagic transformation in cerebral ischemia. Mol Neurobiol 28: 229–244

50. Szabo C (2005) Mechanisms of cell necrosis. Crit Care Med 33(12 Suppl): S530–S534

51. Allport JR, Ding H, Collins T, Gerritsen ME, Luscinskas FW (1997) Endothelial-dependent mechanisms regulate leukocyte transmigration: a process involving the proteasome and disruption of the vascular endothelial-cadherin complex at endothelial cell-to-cell junctions. J Exp Med 186: 517–527

52. Bartynski WS, Tan HP, Boardman JF, Shapiro R, Marsh JW (2008) Posterior reversible encephalopathy syndrome after solid organ transplantation. AJNR Am J Neuroradiol 29: 924–930

PROFORMA

NAME :

AGE :

GENDER :

OCCUPATION :

ADDRESS :

PHONE NO :

CLINICAL SPECTRUM :

- ❖ Headache
- ❖ Visual Impairment
- ❖ Seizures
- ❖ Altered Sensorium
- ❖ Focal neurological deficit
- ❖ Fundi
- ❖ Blood Pressure
- ❖ Other system examination

ETIOLOGY :

- Eclampsia - Antepartum

  -Postpartum

- Pre – eclampsia
- Accelerated hypertension
- Renal
  - Renal Parenchymal Disease
  - Reno Vascular Disease
- Vasculitis
- Connective Tissue Disorders
- Dysautonomia-Gullian Barre Syndrome
- Drugs and Toxins
- Others

RADIOLOGICAL SPECTRUM :

Areas Involved

- Posterior white matter lesion
- Anterior white matter lesion
- Posterior and Anterior white matter lesion
- Subcortical lesion
- Cortical lesion

- Subcortical and Cortical lesion
- Vasogenic edema
- Vasogenic edema+areas of diffusion restriction, if any.

❖ Occipital

❖ parietal

❖ temporal

❖ frontal

❖ Cerebellum

❖ Brainstem

BLOOD INVESTIGATIONS:

❖ Complete Blood Count

❖ ESR

❖ CRP

❖ Urea

❖ Creatinine

❖ Sugar

❖ ANA

❖ RA factor

❖ ANCA

❖ Elisa for retrovirus

❖ HBsAg

❖ Anti-HCV antibody

URINE ANALYSIS

- Urine Routine
- Urine Spot Protein: Creatinine ratio
- 24- hr urinary protein

EEG

CXR- PA view

ECG

USG whole abdomen

Renal Artery Doppler

Carotid Doppler

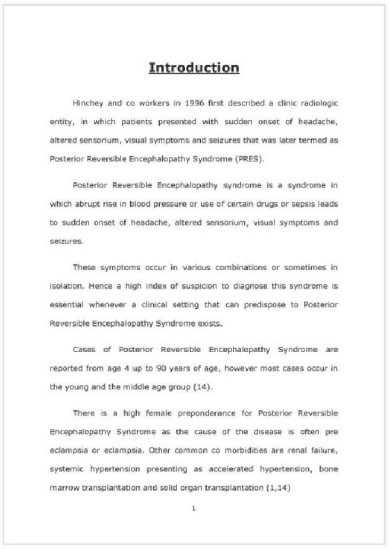
turnitin

Digital Receipt

This receipt acknowledges that Turnitin received your paper. Below you will find the receipt information regarding your submission.

The first page of your submissions is displayed below.

Submission author:	16111051 . D.m. Neurology KALPAN...
Assignment title:	Medical
Submission title:	A study of the clinical spectrum of Pos..
File name:	pages_inPRES.docx
File size:	3.9M
Page count:	52
Word count:	5,466
Character count:	32,817
Submission date:	07-Apr-2014 09:12PM
Submission ID:	414047699



Introduction

Hinchey and co workers in 1996 first described a clinic radiologic entity, in which patients presented with sudden onset of headache, altered sensorium, visual symptoms and seizures that was later termed as Posterior Reversible Encephalopathy Syndrome (PRES).

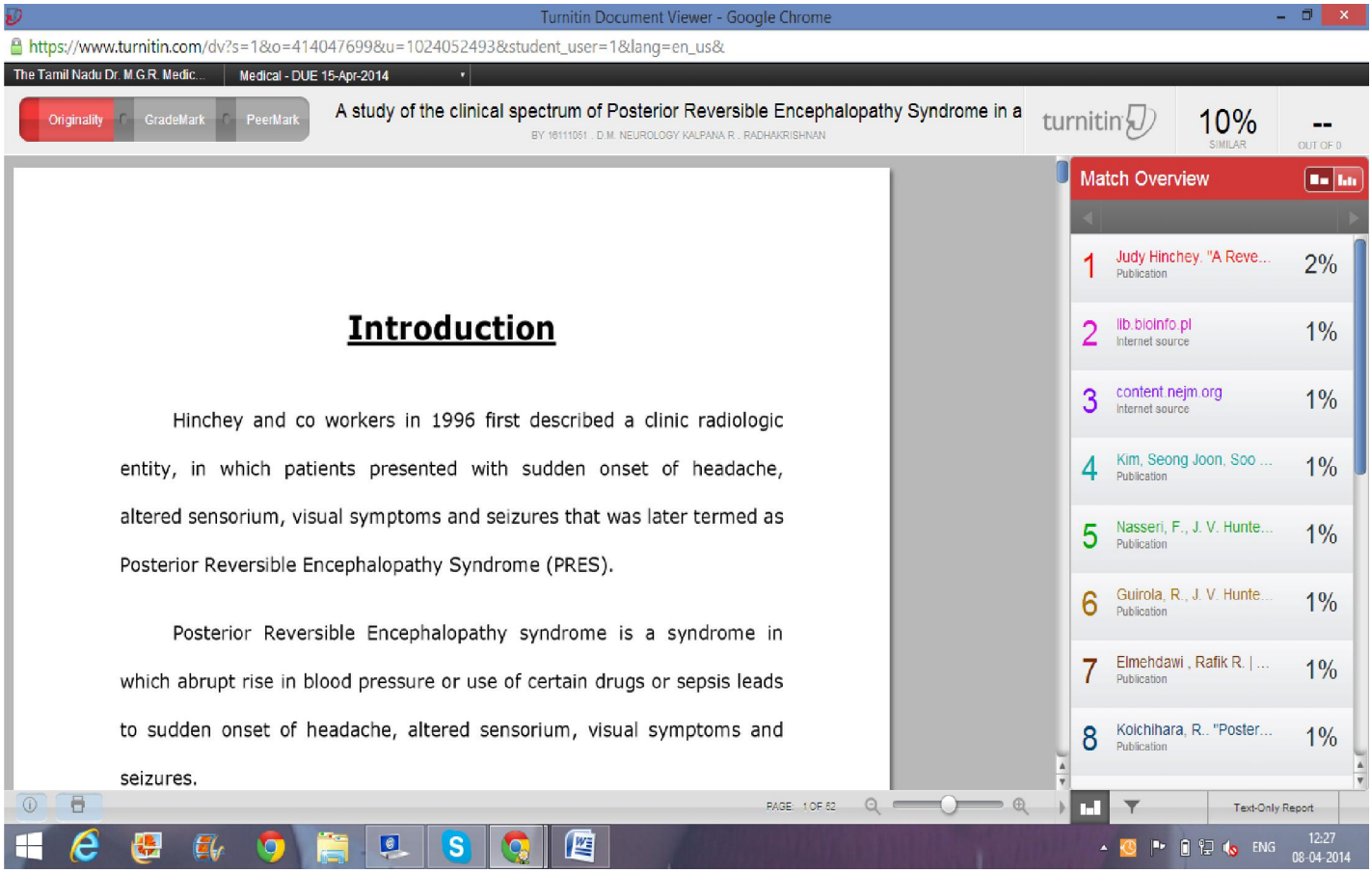
Posterior Reversible Encephalopathy syndrome is a syndrome in which abrupt rise in blood pressure or use of certain drugs or sepsis leads to sudden onset of headache, altered sensorium, visual symptoms and seizures.

These symptoms occur in various combinations or sometimes in isolation. Hence a high index of suspicion to diagnose this syndrome is essential whenever a clinical setting that can predispose to Posterior Reversible Encephalopathy Syndrome exists.

Cases of Posterior Reversible Encephalopathy Syndrome are reported from age 4 up to 90 years of age, however most cases occur in the young and the middle age group (14).

There is a high female preponderance for Posterior Reversible Encephalopathy Syndrome as the cause of the disease is often pre eclampsia or eclampsia. Other common co morbidities are renal failure, systemic hypertension presenting as accelerated hypertension, bone marrow transplantation and solid organ transplantation (1,14)

1


Turnitin Document Viewer - Google Chrome
https://www.turnitin.com/dv?s=1&o=414047699&u=1024052493&student_user=1&lang=en_us&
The Tamil Nadu Dr. M.G.R. Medic...
Medical - DUE 15-Apr-2014
Originality
GradeMark
PeerMark
A study of the clinical spectrum of Posterior Reversible Encephalopathy Syndrome in a
BY 16111051 , D.M. NEUROLOGY KALPANA R . RADHAKRISHNAN
turnitin
10%
SIMILAR
--
OUT OF 0
Introduction
Hinchey and co workers in 1996 first described a clinic radiologic entity, in which patients presented with sudden onset of headache, altered sensorium, visual symptoms and seizures that was later termed as Posterior Reversible Encephalopathy Syndrome (PRES).
Posterior Reversible Encephalopathy syndrome is a syndrome in which abrupt rise in blood pressure or use of certain drugs or sepsis leads to sudden onset of headache, altered sensorium, visual symptoms and seizures.
PAGE 1 OF 62
Match Overview
1 Judy Hinchey. "A Reve... Publication 2%
2 lib.bioinfo.pl Internet source 1%
3 content.nejm.org Internet source 1%
4 Kim, Seong Joon, Soo ... Publication 1%
5 Nasseri, F., J. V. Hunte... Publication 1%
6 Guirola, R., J. V. Hunte... Publication 1%
7 Elmehdawi , Rafik R. | ... Publication 1%
8 Koichihara, R.. "Poster... Publication 1%
Text-Only Report
ENG
12:27
08-04-2014

MASTER CHART-CLINICAL SPECTRUM OF PATIENTS WITH PRES- PART 2

S No	Name	MRI Findings													Final Diganosis
		Lobes					VE	DR	Hemorrhage	Subcortical	Cortical	Subcortical and Cortical	Symmetrical	Asymmetrical	
		O	P	T	F	Others									
1	Kudiya Devi	Y	Y	N	N	N	Y	N	N	Y	Y	Y	Y	N	AP eclampsia
2	Jamuna	N	Y	N	N	N	Y	N	N	Y	N	N	N	N	Pre-eclampsia
3	Janaki	Y	N	N	N	N	Y	N	N	Y	N	N	N	N	AP eclampsia
4	Gomathy	Y	N	N	N	N	Y	N	N	Y	N	N	N	N	Pre-eclampsia
5	Radha	Y	Y	Y	N	N	Y	(R) parietal infarct	0	Y	N	N	N	N	Accelerated HTN
6	Amulu	Y	Y	N	N	N	Y	(R) parietal small infarct	N	N	Y	N	Y	N	AP eclampsia
7	Sangeetha	Y (R)	Y	Y	N	N	Y	N	N	Y	N	N	N	Y	PIGN with PRES
8	Kala	N	(R)	Y	Y	B/L Capsulo ganglionic	Y	N	N	Y	N	Y	Y	N	PP-eclampsia
9	Kanika	Y	Y	N	N	N	Y	N	N	Y	N	N	Y	N	Takayasu's arteritas with PRES
10	Prema	Y	N	N	N	N	Y	N	N	Y	N	N	Y	N	PP-eclampsia
11	Jaya	Y	N	N	N	N	Y	N	N	Y	N	N	Y	N	PP-eclampsia
12	Therasa	Y	Y	Y	N	N	Y	B/L occipital infarct	N	Y	Y	Y	Y	N	Accelerated HTN
13	Kavitha	N	N	N	N	N	N	N	N	Y	N	Y	N	N	Pre-eclampsia
14	Angali	Y	Y	N	N	N	N	N	N	Y	N	Y	N	N	PP-eclampsia
15	Ranjith	Y	(R)	Y	Y	N	Y	B/L occipital pole small infarct	N	Y	Y	N	N	Y	Accelerated HTN, Blunt injury abdomen (R) Kidney contussion
16	Suresh	Y	Y	Y	Y	N	Y	N	N	Y	Y	Y	Y	N	PAN with PRES
17	Kousalya	N	Y	Y	N	N	Y	N	N	Y	Y	Y	Y	N	PP eclampsia
18	Loganayaki	N	Y	N	N	N	Y	N	N	Y	Y	Y	Y	N	Acclerated HTN
19	Malar	N	Y	N	N	N	Y	N	N	Y	N	N	Y	N	Pre-eclampsia
20	Kumari	Y	Y	Y	Y	N	Y	N	N	Y	Y	Y	Y	N	SLE with PRES
21	Chandra	Y	Y	Y	Y	N	Y	N	N	Y	Y	Y	Y	N	Acclerated HTN
22	Sankari	Y (R) occipital	Y	Y	N	N	Y	N	N	Y	Y	Y	N	Y	AP eclampsia
23	Durga	Y	Y	Y	N	N	Y	N	N	Y	Y	Y	Y	N	AP eclampsia
24	Ambika	Y	N	Y	N	N	Y	N	N	Y	N	N	Y	N	Pre-eclampsia
25	Vidhya	Y	Y	N	N	N	Y	N	N	Y	N	N	Y	N	OPC poisoning, toxic neuropathy with acute dysautonomia
26	Bhavani	Y (R) occipital	Y	N	N	N	Y	N	N	Y	N	N	N	Y	AP eclampsia
27	Devi	Y	N	N	N	N	Y	N	N	Y	N	N	Y	N	Pre-eclampsia
28	Dinesh	Y	Y	Y	Y	N	Y	N	N	Y	Y	Y	Y	N	porphyria with SIADH, SHT with PRES
29	Valli	Y	Y	Y	N	N	Y	N	N	Y	Y	Y	Y	N	AP eclampsia
30	Arulanathan	Y (R) occipital	N	B/L temporal	N	B/L Capsulo ganglionic, B/L coronoradiata, centrum semiovale	Y	N	N	Y	N	N	Y	N	Malignant HT with PRES
31	Akila	N	Y	n	N	N	Y	N	N	Y	Y	Y	Y	N	ESRD on hemodialysis, recurrent PRES